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THE NATURE OF INHIBITORY PROCESSES WITHIN THE NERVOUS SYSTEM.

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THIS paper consists of three parts—(1) A brief critical review of the present state of opinion as to the nature of inhibitory processes; (2) some considerations that suggest a certain hypothesis as to the nature of such processes in the central nervous system; (3) the description of some experiments that seem to support that hypothesis.

PART I.—CURRENT THEORIES OF INHIBITION.

Prof. Ewald Hering's celebrated doctrine of the processes in living substance[1] has been made by him, by Prof. Max Verworn, and by many other physiologists, the basis of the

[1] First published in 1878 in the papers entitled "Zur Lehre vom Lichtsinne," and further elaborated in the paper "Zur Theorie der Vorgänge in der lebendigen Substanz," *Lotos, Neue Folge*, Bd. ix., Prague, a translation of which appeared in BRAIN, vol. xx.

assimilatory theory of inhibition, and constitutes also the foundation of Hering's still more celebrated and widely-accepted theory of the visual processes. But the fundamental conception of this doctrine would seem to be imperfectly understood by many of those who accept the theories based upon it, and it is necessary, therefore, to describe it briefly before pointing out why it seems to me to be radically vicious. Verworn's exposition of the doctrine[1] is rather more definite and concrete than Hering's, and he introduces certain terms which facilitate discussion.

Verworn proposes the name "biogen" or "biogen-molecule" for the completed molecules of nerve- and muscle-cells that are immediately concerned in the production of the characteristic activities of those cells. A biogen consists of a stable nitrogenous atom-group, linked with non-nitrogenous atom-groups of two principal kinds, the one rich in oxygen, the other rich in oxydisable atoms, chiefly carbon and hydrogen. In any nerve- or muscle-cell biogens are continually undergoing dissimilation and assimilation. Dissimilation consists in the breaking away from the nitrogenous nucleus of the biogen of the loosely-linked non-nitrogenous atom-groups, and the rearrangement of the atoms of these in more stable forms, such as carbonic acid and water. The nitrogenous nucleus, which Verworn names the biogen-remainder (Biogen-reste), has affinities that enable it to take up such atom-groups as are necessary for the reconstitution of the biogen. This process, by which the biogen-remainder attaches loosely to itself atom-groups rich in chemical energy, is "assimilation." In a resting cell, *i.e.*, one withdrawn from all disturbing influences, assimilation and dissimilation go on slowly and at about the same rate. When a stimulus causes increased dissimilation biogen-remainders accumulate in the cell and determine, in virtue of their affinity for atom-groups supplied by the nutritive fluids, an increase of assimilation. It is assumed that in a similar way a stimulus may cause directly an increase of assimilation and indirectly, through the accumu-

[1] In the "Allgemeine Physiologie," Jena, 1901, and in "Die sogenannte Hypnose der Thiere," Jena, 1898.

lation of unstable biogens, a secondary increase of dissimilation. In either case, if the stimulus be long continued, the secondary process will become equal in intensity to the primarily accelerated process, so that a low-level or high-level equilibrium or equivalence of the two processes will result. This inherent tendency to equivalence of the two processes constitutes the self-regulation of metabolism. "The relation of assimilation to dissimilation in unit of time, which we may express by the fraction $\frac{A}{D}$, and which for the sake of brevity we may designate by the term "biotonus," is of fundamental significance for the most various phenomena of life. It is the oscillations in the magnitude of the fraction $\frac{A}{D}$ which produce all changes in the vital manifestations of every organism."[1]

This doctrine asserts, then, that the extent and vigour of contraction of a muscle-fibre are determined, not by the number of biogens that undergo dissimilation, but by the excess of dissimilation over assimilation. It asserts in fact that in a cell in which increased dissimilation produces certain vital manifestations, these are in some way prevented if at the same time there occurs an equivalent increase of assimilation, and that they are proportionally diminished by any smaller increase of assimilation.

Both Hering and Verworn tell us very explicitly that the two processes, assimilation and dissimilation, go on simultaneously in the same cell or mass-unit of living substance, and that neither one diminishes or in any way prevents the other, but that either one merely tends indirectly to accelerate the other. Yet simultaneously-occurring assimilation is supposed to prevent in some way the vital manifestations which in its absence result from dissimilation. The result of dissimilation is admittedly the transformation of the potential chemical energy of the loosely-linked atom-groups of the biogen into living energy, heat and molar motion in the case of muscle. We are asked, then, to believe that assimilation prevents the appearance of this living energy. *But we know that it does not and cannot do so,* for we know

[1] Verworn, "Allgemeine Physiologie," S. 512.

that the total result of a cycle of metabolism, involving equal amounts of assimilation and dissimilation, is the conversion of a definite quantity of the chemical energy of the food into living energy, and that this is the purpose of the whole process. And we know that the amount of energy so converted from the potential to the living form in unit of mass of living substance and in unit of time is a function of the number of "biogens" dissimilated, and that, by the terms of the theory itself, no amount of simultaneously occurring assimilation can diminish the rate of dissimilation and the amount of energy converted from the potential to the active forms in unit of time. This is the fundamental fallacy that underlies Hering's theory of inhibition, as well as every part of his theory of the visual processes. For the theory of inhibition of Hering and Verworn is simply the assumption that inhibitory stimuli act by causing increase of assimilation, and so preventing in some utterly mysterious and, in fact, miraculous manner the liberation of energy which results from dissimilation, and is the specific vital manifestation of the substance of muscle and nerve.

Hering and Verworn assign, in short, as the cause of the vital manifestations of muscle and nerve, not a real physical or physico-chemical process or processes, such as the cause of all physical effects must be, but a mathematical relation between the intensities of two independent processes. This fallacy, which is so obvious and undeniable when once pointed out, is the fundamental conception of Hering's theory of the processes in living substance, and it is therefore unnecessary to dwell upon the great difficulties that arise when it is attempted to apply it to the explanation of the phenomena of nerve and muscle. I will briefly mention only two of them. Firstly, the theory of inhibition founded on this doctrine necessarily assumes that the assimilatory process is capable of propagating itself along a nerve-fibre. Now, while the propagation of the dissimilatory process is a conception that presents no difficulties, being one for which we have numerous physical analogies in the propagation of chemical processes that

result in the liberation of energy, *e.g.*, the firing of a train of gunpowder, the assimilatory process is in a very different case. For the transmission of such a process, by which no energy is liberated, we can find no physical analogy, and I do not think that it is possible to conceive how the mere linking together of atom-groups without liberation of energy (if, in fact, such a process is at all possible) can determine the occurrence of a similar process among neighbouring molecules. And it may be added that the determination of increased assimilation by any physical stimulus is a process very difficult or impossible to conceive, and one for which we have no physical analogy, that assimilation itself is a process that we do not understand in the same sense that we may claim to understand dissimilation, for in it is involved the essential mystery of life.

A second example of the apparently insuperable difficulties in the way of the application of this theory is the fact of the production of tonic or long-continued activity of nerve and muscle by long-continued stimulation. This is the particular case to which Verworn applies the theory in his work on the pseudo-hypnosis of animals. In these cases we see long-continued stimulation of sensory nerves resulting in long-continued contraction of certain muscle-groups, through a continued transmission of the excitation through certain reflex nervous arcs. Verworn admits that according to the theory the constant stimulus must produce in the reflex arcs a condition of low-level equilibrium or equivalence of assimilation and dissimilation, but he points out that there must exist in the living substance an excess of biogen-remainders, and he assigns the presence of this excess of broken-down molecules in the substance of the nerves as the cause of the continued contraction of the muscles.[1] That is to say, we are asked to believe that the excitement of the sensory organ is continuously transmitted to the muscles, not through the propagation of any physical or chemical change through the nervous arcs, but merely by the fact of the continued presence of an excess of inert

[1] "Sogenannte Hypnose d. Thiere" S. 79.

atom-groups. Surely a case of effect without cause, of reaction without action.[1]

Prof. Wundt proposed in the first edition of the "Physiologische Psychologie," and still maintains in the recently-published fifth edition, a theory of nervous inhibition that is very similar to Hering's, although the terms he uses are different. The only important difference is that, whereas Hering leaves entirely unexplained the way in which the constructive metabolic process is supposed to prevent the manifestations of the effects of the destructive process, Wundt explicitly asserts that it does so by rendering latent again the energy set free by the latter process. But nothing in physiology is more certain than that the completed cycle of the metabolism of muscle has for a result the liberation of a large excess of energy from the potential chemical energy of the food supplied to the muscle, and that, therefore, the constructive processes, if they render latent any energy, can so render latent only a much smaller quantity of energy than is set free by an equivalent amount of the destructive processes. Wundt's theory of the inhibitory processes is therefore equally fallacious with Hering's.

Dr. Gaskell has put forward, independently of Hering and Wundt, an anabolic theory of inhibition. The main points are presented in his article on "The Contraction of Cardiac Muscle," in Prof. Schäfer's "Text-book of Physiology."[2] Gaskell points out that the heart and blood-vessels of vertebrates, the adductor muscle of the claw of the crayfish and of the shell of Anodon, as well as certain other muscles, have been shown to be supplied with separate augmentor and inhibitory nerves, and he suggests that possibly all muscles have such a double nerve-supply. It must be admitted that Gaskell seems to have proved that the true vagus-fibres of the heart exert upon it a direct inhibitory action, but that they do so by promoting anabolism, as

[1] In the recently published booklet "Die Biogenhypothese" (Jena 1903), Verworn makes but little mention of the doctrine of "biotonus," and it would seem that he is prepared tacitly to abandon it. If this be the case it is much to be desired that Prof. Verworn should explicitly avow his change of opinion, in view of the great authority that attaches to his name and of the far-reaching influence exerted by Prof. Hering's theories.

[2] Vol. ii., London, 1900.

Gaskell suggests, is by no means proved. The evidence on which Gaskell chiefly relies in making this suggestion is, firstly, the fact that the heart, after a period of inhibition by vagus-stimulation, beats more vigorously than before; secondly, the fact that, during stimulation of the vagus-fibres going to an injured tortoise-heart, the demarcation-current from the heart undergoes a positive variation, *i.e.*, a variation opposite in character to that produced by stimulation of the augmentor nerve. I suggest with diffidence that if the vagus-stimulation merely checks in some way the rate of katabolism of the heart-muscle these effects will follow, although the rate of anabolism be quite unaltered; for if the degree of fatigue and the electrical changes are affected in an opposite manner by the two processes, a diminution of katabolism must produce on them the same effect as an increase of anabolism. And it appears that Gaskell supposes increased anabolism to inhibit the vital manifestations, the effects of katabolism, only by checking the rate of katabolism, for he writes: "On the other hand, it is conceivable that a sudden increase in the anabolic process would manifest itself for the time being as a diminution of the katabolic process, and so bring about the diminished contraction and other inhibitory effects, although at the same time the net result would be an increase in that process."[1] Gaskell, then, avoids the fallacy into which Hering and Verworn have fallen, for it seems that for him inhibition is essentially a checking of the rate of katabolism brought about indirectly and in some unexplained manner by the increased rate of anabolism. This anabolic theory differs, therefore, radically from Hering's and is at least a tenable one. I merely wish to point out that it is difficult to see that anything is gained by making the assumption that increased anabolism mediates between the inhibitory stimulus and its effects, the checking of katabolism, since the facts seem to be equally well explained by the simpler assumption that katabolism is directly checked by the inhibitory nerve. That such direct checking of katabolism is the essential

[1] *Op. cit.*, p. 219.

nature of inhibition in both muscle and nerve seems to be the view somewhat vaguely held by many, perhaps most, physiologists. I shall discuss it briefly in the following section of this paper.

Another view of the nature of inhibition within the nervous system which must be mentioned is that inhibition consists in the blocking of the paths of discharge of "nerve-centres," the resistance of such paths being raised above the normal in some way. This is the view taken by Dr. Mercier in his paper on "Inhibition," [1] published in this journal in 1889, and by Prof. Münsterberg in the sketch of his "Aktions-Theorie," which concludes his recent volume.[2] It is extremely difficult, if not impossible, to imagine any mechanism by which such blocking of efferent channels could be brought about, and neither author has attempted to suggest any such mechanism. The conception is not only extremely vague and obscure, but is open to all the objections that I have to urge against the view that inhibition within the nervous system is due to direct checking of the katabolism of nerves or nerve-centres.

It is of historical interest to note that the oldest theory of nervous inhibition, that of A. W. Wolkmann,[3] proposed to regard inhibition as a process of interference of waves of nervous action in a manner analogous to the interference of sound and light waves. This suggestion has been occasionally repeated, but hardly seems to call for serious consideration.

Part II.—The Hypothesis of Inhibition by Drainage.

The above review of the various theories of inhibition seems to leave only two competitors in the field: (1) inhibition may consist in the direct checking of katabolism; (2) inhibition may consist in the cutting off of nervous influences that cause increased katabolism. The latter view was for a long time very generally accepted as true for all

[1] Brain, vol. xi.
[2] "Grundzüge der Psychologie," Leipzig, 1900.
[3] Wagner's "Handwörterbuch d. Physiologie," Bd. 2, Brunswick, 1846.

kinds of inhibition outside the nervous system. But since Gaskell and others have shown that the heart and other of the visceral muscles have an intrinsic power of contraction, rhythmic or continued, and since Gaskell has proved that the true vagus-fibres exert an inhibitory action directly upon the heart muscle, it must be admitted that in the case of these visceral muscles inhibitory nerves may act by checking the rate of katabolism. If nerve can act thus upon muscle it is probable that it may act upon nerve substance in a similar way, and the problem before us is—Do both, or does only one, of these two kinds of inhibition occur in the nervous system?

In seeking the answer to this question we may gain some indications from certain general considerations. In the first place it seems to be proved that inhibition of skeletal muscles in vertebrates consists always in a cutting off or diminution within the nervous system of the excitatory impulses issuing to the muscles along the motor nerves, and that there is no evidence either of special inhibitory nerve-fibres or of the passage of inhibitory impulses along the motor nerves. This was the conclusion at which Dr. Waller arrived in 1892,[1] and the question seems to have been definitely settled in this sense by a recent research of Verworn.[2]

We have, then, the following state of affairs: Inhibition of muscles by special inhibitory nerves seems to occur among various invertebrata, and to be the rule in the case of the muscles of the vascular and visceral systems of the vertebrates, whereas inhibition of the skeletal muscles of vertebrates is effected by the cutting off within the central nervous system of excitatory impulses passing to the motor nerves. Now the vascular and visceral musculature of vertebrates may undoubtedly be regarded as of a more primitive type than the skeletal musculature.[3] And it is

[1] BRAIN, vol. xv., "On the Inhibition of Voluntarily and of Electrically Excited Muscular Contractions."

[2] "Zur Physiologie der nervösen Hemmungserscheinungen," *Arch. f. Anat. u. Phys.*, 1900.

[3] This interesting aspect of the question was pointed out to me by Prof. Gotch.

obvious that inhibition by special nerves or impulses sent to the muscles would be a clumsy and wasteful method of controlling the muscles if the same end can be achieved by a simple cutting off of the excitatory impulses. There is, then, good reason to regard inhibition by special inhibitory nerves, as we see it in the crayfish and in the bivalve, as a type of physiological mechanism that has been tried by Nature and found wanting, or, at least, as one that has been superseded in the case of the skeletal muscles of the higher animals by a principle of superior economy and efficiency.

On the other hand, we may note that all those muscles of the visceral system of the vertebrate that are supplied with special inhibitory nerves exhibit a considerable power of spontaneous activity, a power that is lacking in the skeletal muscles. While, then, inhibitory impulses are necessary if the nervous system is to control the activity of these visceral muscles, no such impulses are needed in the case of the skeletal muscles in which spontaneous activity is lacking or at a minimum.

But these two ways of looking at the problem reveal but two aspects of the one truth. The lack of spontaneity of the skeletal muscles of vertebrates and the corresponding absence of inhibitory nerves or impulses must be regarded as results of that increasing integration of the whole organism, that increasing centralisation of control in the brain, which is the most striking feature of the evolution of the higher types of animal. In the more primitive segmented types the individuality of the animal is very imperfect. But the higher we rise in the scale of animal life the more do we find the spontaneity of the parts giving place to a dependence upon the central nervous system, and especially the brain, for the initiation of their activities; that is to say, the activities of the skeletal muscles are brought into a more and more intimate relation with the environment through the sense-organs and reflex nervous arcs, while the viscera remain comparatively independent of such control, retaining in a great degree their power of spontaneous movement.

Since then we find that, while inhibition of visceral

muscles is effected through special nerves, inhibition of the skeletal muscles is effected more simply by the cutting off of the outflow of excitatory impulses to them, we may fairly expect to find a similar difference in the mechanism of inhibition throughout the parts of the nervous system controlling the visceral and the skeletal musculature respectively. And in man and the higher vertebrates the part of the nervous system that controls the skeletal muscles constitutes by far the larger part of the brain and probably the larger part of the spinal cord also. It may well be, therefore, that the more primitive inhibitory mechanism persists in the small part of the nervous system that directly controls the visceral (including the respiratory) functions, while in the rest of the central nervous system we may expect to find inhibition effected simply by the cutting off or withdrawal of excitatory influences.

This indication is borne out by other considerations. If inhibition is effected by special inhibitory impulses, one of two hypotheses, or both, must be true—either the inhibitory impulses are carried by special nerves, or they are carried by the nerves that carry also excitatory impulses. The former hypothesis we may reject at once with confidence, for all parts of the nervous system are so connected that excitement may spread from any one part to any other, and further, all parts are so connected that excitement of any one part may inhibit the activity of any other, as when some violent sensory stimulus arrests all perception, thought and movement, both voluntary, automatic, and reflex. If we assume that inhibition is brought about through special nerve-fibres we shall then have to suppose that all parts are interconnected by a duplicate set of paths, the excitatory and the inhibitory, that, in fact, almost the whole central nervous system exists in duplicate. Such an anatomical basis for the inhibitory processes would be a very clumsy and complicated affair, and not only is its existence, therefore, extremely improbable, but it is impossible that, if it existed, we should not have obtained some direct evidence of the fact.

If, then, we believe that inhibition is effected by special

inhibitory impulses, we must believe that all or almost all nerve-fibres in the central nervous system are capable of carrying two sorts of impulses, the excitatory and the inhibitory. And this would seem to be the view held by most of the physiologists who do not accept Hering's assimilatory theory. But there are grave objections to this view. In the first place, it has not been proved that an inhibitory impulse, as something different from and opposite in character to an excitatory impulse, is conveyed by any nerve whatsoever. For although Gaskell's work seems to prove that the vagus fibres inhibit the heart by acting directly upon the muscle-substance, it remains open to us to believe that the impulses conveyed by the excitatory and inhibitory nerves are essentially similar in character, and that the difference of their effects is due simply to a difference in the mode of connexion of the two sets of fibres with the muscle-substance (*e.g.*, it may well be that while the one set is directly connected with the sarcostyles, the other set is connected with the sarcoplasm only). And there exists experimental evidence in favour of this view.

If we postulate two kinds of impulse in nerve-fibres, we must believe that either kind preserves its type in transmission across synapses from neurone to neurone. Now Professor Langley has recently shown[1] that the central end of the cut vagus, or of the lingual nerve, may be joined to the peripheral end of the cut cervical sympathetic below, *i.e.*, on the proximal side of, the superior cervical ganglion, and that under these conditions there may occur a remarkable restoration of the functions normally dependent on the activity of the cervical sympathetic nerve; and Langley writes: "It seems to me that the evidence strongly favours the view that the *vaso-dilator fibres* of the lingual (chorda tympani) after becoming connected with the nerve-cells of the superior cervical ganglion, become *motor fibres* for unstriated muscle, and in especial *vaso-constrictor fibres* for the arteries of the ear." And Langley adds: "I have earlier brought forward some facts to show that the function

[1] "Regeneration of Autonomic Nerves," *Journal of Physiology*, vol. xxiii.

of the peripheral nerve-cell depends upon the peripheral structure in which its axon has an opportunity of ending. So that the physiological differences depend, in the main, upon the anatomical connections brought about by morphological laws." We have, then, the remarkable fact that an inhibitory nerve-fibre will make junction by synapse with an augmentor fibre leading to a muscle, and that stimulation of the inhibitory nerve then causes contraction of the muscle. And Langley describes also the converse of this case, the production of inhibition by stimulation of an augmentor nerve that has made junction with an inhibitory nerve.

If these cases do not suffice to prove that the impulses conveyed by augmentor and by inhibitory nerves are similar in character, they yet lend this view a very strong probability. And we may fairly assert that there is no conclusive evidence that there is any essential difference of character between the impulses conveyed by nerves of different function, and that, in fact, all the available evidence points the other way. Still less has it been shown that any one nerve is capable of conducting impulses of more than one kind. The very fact that in the case of the visceral muscles, for the control of which inhibitory impulses are necessary, the inhibition is effected by special nerve-fibres and not by the fibres that carry excitatory impulses to the muscles, this fact affords a strong presumption that it is not possible for a nerve to carry two kinds of impulse. We must, therefore, hesitate long before we make the great assumption that all or most nerve-fibres carry impulses of two opposite kinds, augmentor and inhibitory, and if we can find an hypothesis that does not involve this large and unbased assumption we must regard it as, in this respect at least, highly preferable.

But if, overlooking this primary objection, we make this assumption of two kinds of conduction-processes in all nerve-fibres, serious difficulties arise when we attempt to apply the hypothesis to the explanation of inhibitory phenomena. In the first place we know that inhibitory effects may spread widely, and we shall have to assume that either kind of conduction process is capable of preserving its type in

passing across a synapse from neurone to neurone, and this is difficult to conceive. For we know that in transmission across a synapse the excitation-process suffers profound changes, that it is delayed and may suffer a change of rhythm, and there is good reason to think that the transmission across the synapse involves a material substratum highly peculiar in character.[1]

A still more serious difficulty is revealed by the following considerations. It seems to be a rule, with but few exceptions, that a stimulus applied to one afferent tract produces excitation in some parts and inhibition in other parts of the central nervous system. We have, then, to imagine that an excitation-process initiated in one part may spread as excitation through one tract or tracts while it spreads as an inhibitory process through other tracts; we shall have to imagine, in fact, a system of stations mediating between different tracts, in which stations the impulse suffers a conversion from one type to the opposite type. It would be possible to imagine such a system of converting stations,[2] but for the fact that the relation between neurones and systems of neurones, as regards inhibition and excitation, is a highly variable one. Under one set of conditions excitation of one system of neurones may cause inhibition of a second system, while under other conditions it may spread as excitation throughout that second system. Thus Sherrington writes: "In the spinal animal, inhibition due to excitation of a nerve-path or centre is usually quite transient, and passes over into excitation even during the continuance of the stimulation if that be fairly prolonged."[3] Such variability of the relations between nerve-tracts as regards excitation and inhibition seems to show that we may not assume that, when excitation of one tract causes inhibition

[1] The evidence for this is set out in my paper, "On the Seat of the Psycho-Physical Processes," BRAIN, 1901.

[2] Wundt has imagined such a system of converting stations. He supposes that the cell-body of each neurone consists of two parts, a central part in which constructive metabolism predominates, a peripheral part in which destructive metabolism predominates, and he supposes that, according as a nerve-fibre makes connection with the central or peripheral part, it will initiate in that neurone an inhibitory or an augmentor process.

[3] Art. "Spinal Cord," Schäfer's "Textbook of Physiology," p. 841.

of another, it is because the conduction-process suffers a conversion of type in some apparatus mediating between the two systems; and if we cannot assume the existence of such converting stations we cannot conceive that any such conversion of the type of process is possible.

We find ourselves, therefore, driven to adopt the view that inhibition in the greater part of the nervous system, all the part concerned in the control of the skeletal musculature, consists simply in the cutting off from the tract inhibited of the excitatory impulses by which alone its activity can be maintained. When we seek an hypothesis as to the mechanism by which this cutting off of excitatory influences may be effected we find that clear and unmistakable indications are afforded by certain psychological considerations.

Inhibition in the Highest Levels of the Brain.

The mind can attend to only one object at one moment of time,[1] and this singleness of the attention is clearly a mental characteristic of supreme importance to the individual in the struggle for existence. For the singleness of the attention secures the unity of the individual as expressed in action; the object or idea, the presentation to which attention is given at any moment, is the principal factor in determining conduct at that moment, and if a man could attend to more than one object at one moment he would be like a house divided against itself. Different objects would call simultaneously for different, and often antagonistic, movements of parts of, or of the whole of, the body; the unfortunate man would frequently find himself in the situation of the mythical ass that, standing between two bundles of

[1] Prof. W. James writes ("Principles of Psychology," vol. i., p. 405): "The number of *things* we may attend to is altogether indefinite, depending on the power of the individual intellect, on the form of the apprehension, and on what the things are. When apprehended conceptually as a connected system, their number may be very large. But however numerous the things, they can only be known in a single pulse of consciousness for which they form one complex 'object,' so that properly speaking there is before the mind at no time a plurality of *ideas*, properly so called." Any reader who may be inclined to dispute the singleness of the attention may be referred to Mr. F. H. Bradley's recent article on "Attention," *Mind*, No. 41, 1902.

hay, could not make up its mind to turn away from one in order to eat the other. Such a situation is never realised in man or animals because our attention is single; and although in such a situation the attention may oscillate for a time between two attractive objects, one or other of them soon fixes the attention sufficiently to determine action.

The turning of the attention (voluntarily or involuntarily) from object *a* to object *b* (*e.g.*, from a visual to an auditory presentation, in consequence of a sudden increase of the intensity of the latter) while both continue to affect the sense-organs, means that the excitation-processes initiated by *b* have suddenly attained a greater range, have penetrated further into the higher levels of the brain, while those initiated by *a* have as suddenly ceased to penetrate to systems of neurones in the same higher levels, and have become restricted to neurones of the reflex and sensory-reflex levels only.[1] The wider extension of the process initiated by *b* has resulted in a restriction of the spread of the process initiated by *a*. In a similar way any sensory presentation or idea in becoming the object of attention, the central object of mental activity, causes any other object or idea to cease to be attended to. It is, therefore, clear that there obtains a relation of reciprocal inhibition between all those numerous organised systems of neurones or nerve-paths, the physiological bases of the mental systems, which have been built up through successive apperceptions (partly of the individual, partly of the race) to bring about appropriate reactions to every kind of object presented to the senses, or, as we say when the reaction is complex, appropriate conduct in relation to the object. Further, it would seem that we have no power of simply ceasing to attend to any object or idea, but that we can withdraw our attention from one object only by giving it to some other. This fact was very clearly expressed by Edmund Gurney in combating the statement that we are capable of simply and directly withdrawing the attention from any object. He wrote: "This direct action of inhibition is hard to picture. This

[1] See my article "Physiological factors of Attention-process," *Mind*, No. 43.

activity of active inhibition appears to me always to lie in a determined setting of the mind in some new direction. I mitigate a pain, not by attending either to the pain or to an imagined absence of pain, but by clenching my teeth and thinking of something else, *i.e.*, by opening quite new channels of nervous energy."[1]

It appears, in fact, that the inhibition of a mental process is always the result of the setting in of some other mental process, and, if we consider the underlying physiological processes, we see that this means that the inhibition of the excitation of one neural system is always the result of the excitement of some other system, *that inhibition appears always as the negative or complementary result of a process of increased excitation in some other part.* This fact suggests that inhibition is essentially the result of a process of competition, and many psychologists have given expression to this conception in some such vague phrase as: The mind has only a limited quantity of energy, which will not suffice for the simultaneous maintenance of two mental processes.

But as to the mechanism of the process of competition we have hardly any, even the vaguest, suggestion. It is, of course, possible to hold, and by the older psychologists it was commonly held, that the singleness of attention is due to the soul, it being postulated that the soul or ego is by nature single or simple and therefore incapable of entertaining more than one idea at any moment; or the soul was likened to the retina, which possesses a central spot of clearest vision surrounded by zones of vision of diminishing clearness, and it was supposed that only that nervous excitation-process, which for some unknown reason succeeded in affecting this central or focal point of the soul, could determine clear and attentive consciousness. Such speculation sounds somewhat antiquated to modern ears, but Lotze, who represented the flower of physiological and psychological learning of his time, wrote just fifty years ago: "The soul is not, indeed, so organised as to know all

[1] "The Problems of Hypnotism," *Mind*, O.S., vol. ix.

its inner conditions with the same degree of clearness and attention; it resembles rather the retina of the eye, in which a larger area of symmetrically decreasing excitability surrounds a single spot of clear sensibility."[1]

But the tendency of modern thought compels us to seek a physiological basis for the singleness of attention.[2] Accordingly we find that several physiological-psychologists foreshadow more or less vaguely the hypothesis that inhibition is due to competition among nerve-paths for the living energy present at the moment in the nervous system. The most definite presentation of this conception that I have read is contained in those most interesting pages which conclude Prof. James' chapter on the Will,[3] in which he writes of inhibition through drainage of one cell by another.

There are many facts which lead us to believe that the living energy in the nervous system, or, more particularly, in the afferent side of the nervous system (including a large part of the cerebellum) at any moment forms, in a sense, a common store or reservoir of energy upon which the different paths leading to different muscle groups can draw. I have recently put together the more important of these facts in a paper,[4] which will appear shortly, and to this I must refer the reader for the justification of this conception. But lest it should be thought that I am talking wildly and without due regard to the opinions of physiologists, I will quote the following passages from a recent utterance[5] by Dr. F. W. Mott, one of our highest authorities on the nervous system. He writes that stimulation may spread in the gray matter "and cause the liberation of stored energy,

[1] *Medicinische Psychologie*, p. 505.

[2] It is interesting to note that if the attempt to find a physiological basis for the singleness of attention should prove successful, it will be open to us to suppose that, given a different type of physiological mechanism, the soul might be capable of attending simultaneously to an indefinitely large number of ideas or objects. In cases of double personality, and in some cases of automatic writing and speaking, we seem to have instances of multiple activity of the soul owing to disorganisation and loss of functional unity in the nervous system.

[3] "Principles of Psychology," vol. ii., p. 579, *et seq.*

[4] "Physiological Factors of the Attention-Process," p. ii., *Mind*, July, 1903.

[5] "The Importance of Stimuli in Repair and Decay of the Nervous System," *Journal of Mental Science*, October, 1902. The italics are mine.

or *redistribution of active energy*"; and, again, "Currents which represent nervous energy are continually flowing in all directions in the central nervous system. They flow with the greatest readiness along systems of neurones which have by habit and use been most functionally correlated." And again he writes: "If we suppose that the total nervous potential (stored nervous energy) is *at the disposal of the whole nervous system*, then in the insane we must suppose that it is constantly being used up in a wrong way"; and again, "For attention is both a positive and negative process as regards stimulus. We cannot concentrate consciousness upon stimuli from some external object without shutting out of consciousness all other stimuli. *Is this a process of switching off as well as switching on of active potential*, or is the negative effect the result of opposition of nervous currents?" In these passages Mott suggests, and in part adopts, just those conceptions for which I am contending, namely, (1) the distinction of the potential and living energy within the nervous system at any moment. It is because I feel so strongly the need for a clear conception of the living energy as distinguished from the chemical potential energy of the nervous system that I have suggested the name "neurin" in place of the cumbrous phrase "living nervous energy,"[1] believing that to give a distinctive name to any conception is an important step towards rendering it clear and definite and useful; (2) that the living energy flows in currents from part to part, and in so doing follows the paths of least resistance; (3) the fact that the energy of the nervous system as a whole is more or less at the disposal of every part; (4) that inhibition means a switching off of the current of energy or neurin, and that a movement of attention means the switching off of the current from one path of forward-conduction in the higher levels of the brain and the turning of it into some other similar path which, through a complex constellation of influences, has become at that moment the path of least resistance in those higher levels. The relation of reciprocal inhibition obtaining between all

[1] "On the Seat of the Psycho-Physical Processes," BRAIN, Winter, 1901.

the different organised neural systems, constituting paths of forward-conduction in the higher brain-levels, is due, I suggest, to the fact that they all drain one common store of neurin contained and constantly generated in the interconnected mass of afferent neurones, and seeking constantly to escape by the paths of least resistance into motor neurones, and so into the muscles. The higher-level paths are brought into activity only when the store of neurin attains a certain potential or head of pressure, which degree of pressure is an essential condition of attentive consciousness. And only one of these higher-level paths can be active at any one moment, because any one of them is capable of carrying off the whole surplus of neurin; when, then, any combination of causes reduces the resistance of any other path to a lower point than that of the path active at any moment, the current shifts from one to the other, just as the opening of a new and shorter channel for a river causes the stream to flow wholly in the new channel, leaving in the old river bed only stagnant pools of water.[1]

This view of the nature of inhibition as it occurs in the higher brain-levels is, I believe, not only tenable, but at the present time the only tenable one. It is the only hypothesis that enables us to understand, however imperfectly, the universality of the relation of reciprocal inhibition obtaining between all the multitudinous systems of paths in those higher levels.

The study of inhibition as it occurs in the highest levels of the brain suggests, then, a certain view of the nature of the process as the only tenable one. Now, we regard these higher levels as a late outgrowth from, and complication of, the lower parts of the central nervous system, evolved in the course of ages to effect an ever-increasing nicety and complexity of adaptation of movements to sensory impressions, and we may, therefore, expect to find the same fundamental principles underlying the activity of all the parts of the nervous system concerned in this work. If, then, this

[1] We can only very roughly picture the marvellously delicate and complicated play of influences bringing about this perpetual shifting of the main current of energy from one neural system to another throughout the hours of waking life.

view is the true one we shall find it to be applicable to other kinds of inhibitory process in the nervous system.

Besides the inhibitions which constitute the negative aspect of the attention-process, we may distinguish two other principal types of inhibition: (1) inhibitions within the sensory-reflex level of one sensory process by another; (2) inhibitions in the purely reflex level, including the most typical cases, the reciprocal inhibitions of the reflex paths innervating antagonistic muscle-groups, the recent demonstration of which by Prof. Sherrington has constituted so great an advance in our knowledge of the physiology of the nervous system.

Sherrington himself has suggested the essential similarity of the process in these two classes.[1]

Inhibition in the Spinal Cord.

Physiologists are agreed that inhibitions in the spinal cord play a very important part in securing the coördination of movements, and though but few general laws as to the conditions which determine inhibition have been established, certain facts seem to stand out prominently:—Firstly, "the solidarity of the spinal cord is great enough for its tonus at any one point to be a function of the whole spinal status—indeed, of the condition of the entire nervous system—to be a function, that is, of the living of other spinal parts, especially of those adjoining to it, as well as of that of the individual part itself";[2] and, "the condition of one part of the cord influences the reactions of the other parts."[3] That is to say, there obtains in the spinal cord a solidarity, a capacity for reciprocal influence of the different parts, comparable to that which we have seen to obtain in the highest

[1] "The condition of one part of the cord influences the reactions of the other parts. In this respect its homogeneity may be likened to that of the retina (simultaneous induction)," Schäfer's "Text-book of Physiology," vol. ii., p. 839; and again, "The local exaltation or depression effected by a real reciprocity in the retina may be likened to the local spinal exaltation and depression I have called attention to in the locality of severed (or conversely excited) sensory spinal roots, with local hyperæsthesia or diminished knee-jerk for criteria."—"Retinal Reciprocal Action," *Journ. of Phys.*, vol. xxi.

[2] Sherrington, "Retinal Reciprocal Action," *Journ. of Phys.*, vol. xxi.

[3] *Loc. cit.*

nervous levels and which appears to be the physiological basis of the singleness of attention. Secondly, excitation of one reflex arc very frequently causes inhibition of the excitation of others, and, as in the case of the higher brain-levels, inhibition of the activity of one arc seems to appear invariably as the negative or complementary process to increased excitation of some other part. Sherrington lays down the rule that sensory stimuli "which tend, when separate, to excite different but inter-related groups of motor cells, tend, when contemporary, to inhibit one another." [1]

Further, "It is probable that inhibition of one spinal 'centre' by another plays a great part in the elementary coördination of actions executed under spinal mechanisms. The augmentor innervation of one muscle-group seems regularly to be accompanied by inhibition of the innervation of another muscle-group, this mode of innervation I have termed 'reciprocal.' It seems a phenomenon common in 'spinal reactions.'" [2]

Of all spinal inhibitions the reciprocal inhibition of antagonistic muscle-groups is the best-defined class and may serve as the type. Sherrington has shown that this relation obtains between numerous pairs of antagonistic muscle-groups, and it seems probable that it obtains between most, if not all, such pairs.

It is easy to suggest a scheme of the connections between two such arcs that will explain how the inhibition of the less-intensely stimulated arc by the more-intensely stimulated arc may be the result of a process of competition. Let us imagine each arc in simple schematic form as a chain of three neurones,[3] afferent (a_1), central (a_2), efferent (a_3), and let us call them a_1, a_2, and a_3, and b_1, b_2, and b_3 in the two arcs respectively. In the resting state each arc maintains a gentle tonic contraction of its muscle-group, its afferent end being constantly subject to gentle stimulation partly by the

[1] Sherrington, Schäfer's "Text-book," p. 842.

[2] The same, *op. cit.* p. 840.

[3] It is asserted that some branches of afferent neurones of the cord make direct connection with motor neurones, while others do so only indirectly by means of an intercalated neurone. *See* Barker, "The Nervous System," p. 952, &c.

muscles themselves, partly, but to a less extent, by stimuli affecting the skin.[1] The synapses of either arc present, in the resting state, a certain degree of resistance determined by the past history of each synapse in the individual and in the species, a resistance that is diminished by the charging of the neurones between which it mediates, and is increased by the fatigue that results from the continued transmission of the impulse. The neurin generated in either arc we may conceive as escaping into the motor neurone through the resistant synapses of the resting arc by leakage.[2]

When a strong stimulus is applied to the afferent neurone of arc a it generates neurin rapidly, so that it becomes very rapidly charged, and the resistance of synapse a_1—a_2 lowered, until a series of discharges takes place from

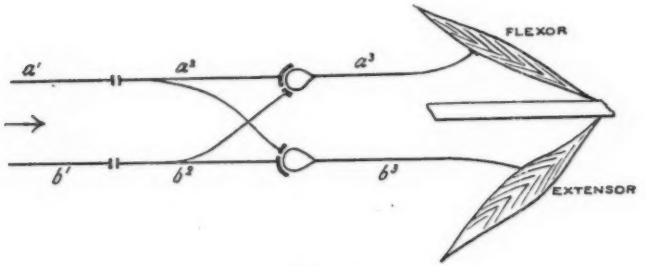

FIG. 1.

a_1 to a_2, and again from a_2 to a_3. The problem then is to imagine such a mode of connection between arc a and arc b as will cause the arc a during stimulation to drain off from the afferent and central neurones of arc b the smaller quantities of neurin generated in them. Several forms of such a connection may be imagined, but I think that probably it takes the form of a collateral fibre coming from neurone b_2, and taking part with the axon of a_2 in forming a synapse with a_3. It may be that this synapse takes the

[1] Sherrington, *op. cit.*, p. 870.

[2] I have displayed in some detail in a former paper the evidence that justifies the conceptions introduced in this scheme. BRAIN, Winter Number, 1901.

form of a basket applied to the body of the motor neurone.[1] But whatever the exact constitution of this synapse may be, we may assume that, when its resistance is lowered by the stimulation of a_1, and consequent charging of a_2, the collateral of b_2, making connection with a_3 through this synapse, becomes the path of least resistance for the escape of neurin from b_1 and b_2. These neurones are therefore drained by a_3, while b_3 ceases to receive any neurin from b_2, and the tone of the muscle-group supplied by it is abolished.

In a similar way, if a collateral from a_2 joins with the axon of b_2 in forming a synapse with a_3, a strong stimulus applied to b_1 will cause charging and discharging of the arc b, with lowering of resistance of synapse b_2—b_3, and consequent drainage of a_2 into b_3, *i.e.*, inhibition of tonus of muscle-group supplied by a_3. In a similar way, if both a_1 and b_1 be stimulated, but one more strongly than the other, the more-strongly stimulated arc will drain the afferent and central neurones of the less-strongly stimulated arc, because the resistance of synapses of the former will be reduced to a lower level than that of the synapses of the latter.

This scheme enables us to understand (1) why it is that inhibition in one part always appears as the negative aspect of excitation in another; (2) the fact that a stimulus to a given spot of skin may cause a reflex contraction of flexors or extensors of a limb according as extensors or flexors have been in tonic reflex contraction for some time before the application of the stimulus[2]; (3) a very perplexing feature of reciprocal inhibition, the fact, namely, that "in the spinal animal, inhibition due to excitation of a nerve-path or centre is usually quite transient, and passes over into exaltation even during the continuance of the stimulation

[1] Our knowledge of the basket-like synapses is still very imperfect. Held, after stating that they have been observed in numbers both in the cord and brain, writes: "Entweder eine hauptsächliche Nervenfaser oder mehrere und zahlreiche sogar können zu dieser korbartigen Hülle der Ganglienzellen zusammenkommen, die somit als eine von Nervenfaser-verzweigungen gebildete reizumleitende Einrichtung aufgefasst werden kann."—H. Held, "Ueber den Bau der grauen und weissen Substanz," *Archiv. f. Anat. u. Phys. Anat. Abth.*, 1902.

[2] Sherrington, *op. cit.*, p. 844.

if that be fairly prolonged"; [1] and that, "the reflex spinal movement, if prolonged, tends always, in my experience, to be of *alternating* character. . . . A movement from the initial posture of rest to another posture is obtained; this is the *primary* movement, and with it the reflex reaction, if of short duration, may come to an end. But with prolongation of the reflex action the posture first attained is not maintained. A second movement, opposed in sense to the former, sets in. And this alternation of postures may be repeated a number of times." "In their phasic alternation of condition the motor-cells pass through periods of discharge and quiescence; the contraction and the relaxation contemporaneously affect antagonistic muscles," and throughout the phases of this sequence "inhibition as well as excitation seems to play a part." [2]

In applying the scheme of fig. 1 to the explanation of this kind of alternating reflex effect we must suppose the collateral connection of the arc a (to which we may suppose the stimulus to be applied) with the neurone b_3 to be but little inferior in conducting capacity to its direct connection with a_3. When, then, the neurone a is stimulated continuously the arc will first discharge into a_3 and drain b_1 and b_2 (*i.e.*, inhibit b_3), until after some little time fatigue causes the resistance of synapse a_2—a_3 to become slightly greater than that of synapse b_2—b_3, when a_2 will discharge with b_2 wholly into b_3, and a_3 will be in turn "inhibited." So the charges of the afferent neurones of both arcs will be discharged into b_3, until fatigue causes rise of resistance of the synapse on b_3, when the discharge will again take place through the now restored synapse on a_3.

Since Sherrington himself has drawn attention to the similarity of these relations of reciprocal inhibition between reflex arcs of the cord to those obtaining between parts of the retino-cerebral tracts, I do not hesitate to point out that we may observe in those tracts an instance of alternating activity and inhibition of two related tracts affected by constant stimulation, and that this instance constitutes an

[1] Sherrington, *op. cit.*, p. 841.
[2] Sherrington, *op. cit.*, p. 830.

extremely close analogy to the cases of alternating inhibition and excitation in spinal arcs. I mean the physiological processes that underly the struggle of two different colours presented to corresponding parts of the two retinæ. In a former paper[1] I have adduced experimental evidence to show that the alternate inhibition and excitation of the two tracts, which occurs under these circumstances, depends upon a fatigue of the tracts, presumably of their synapses, which is rapidly produced and as rapidly recovered from. It only remains to point out that even as regards the duration of periods of alternation the similarity is close. Sherrington gives from three to five seconds as a typical period of duration of either phase in the case of the spinal arcs,[2] and the alternating phases of colour occupy usually very similar periods during moderately intense stimulation.

Such cases of reciprocal inhibition are readily explicable in terms of my hypothesis, whereas it seems very difficult or impossible to explain them if we suppose inhibition to be brought about by special inhibitory impulses checking katabolism; we should have to suppose that a constant stimulus sends excitatory impulses through one tract and inhibitory impulses through another, and that at regular intervals, without any change occurring in the character of the stimulus, the process in either tract undergoes a sudden conversion into its opposite.

Part III.—Some Experiments Supporting the Hypothesis.

I should hardly venture to put forward the above somewhat speculative applications of my hypothesis if I were not able to adduce some direct experimental evidence in support of it. Such evidence is, I believe, supplied by the study of visual sensations. Here I would take the opportunity to urge the importance of the study of sensations for the science of neurology. Apart from necessarily uncertain inferences

[1] Brain, Winter Number, 1901.
[2] *Op. cit.*, p. 832.

from anatomical facts, our knowlege of the physiology of the nervous system depends almost wholly on three classes of observation: (1) observations of the effects produced in muscles by changes in the nervous system; (2) observations of the electrical changes in nerves; (3) the study of sensations. It may well be that the first and second of these methods of attacking the problems of neurology are now nearly worked out, and have already yielded us the chief part of the harvest that they are capable of yielding. On the other hand sensations afford a field of study of inexhaustible variety, and, although they have been the objects of much detailed observation, the correlation of them with the neural processes underlying them is a subject for research that is yet in its earliest infancy, and promises to be of the utmost importance for neurology.

Inhibitions in the Sensory-Reflex Level.

Except in the case of the visual sensations very little is known of the interferences of sensations due to simultaneously applied stimuli. The general rule has been laid down that, "the quality and intensity of a sensation depend not only upon the stimulus which affects one part of the sense-organ, but also on the immediately preceding stimulation of the same part and on the simultaneous stimulation of other parts of the same organ."[1] And there are a good many scattered facts which go far to justify a general statement that of sensations due to simultaneously applied stimuli, the feebler sensation is weakened by the more intense. This inhibition of the weaker by the stronger is especially obvious, and has frequently been noted, in the case of simultaneously caused pain-sensations. The reciprocal modifications of visual sensations have been much studied, and the weakening of the less intense by the more intense sensation appears clearly in a multitude of cases. In a former paper[2] I have shown that all the phenomena

[1] C. Stumpf. "Jonpschologie," Bol. i., S. 19.
[2] "Some New Observations in Support of Young's Theory of Light and Colour-Vision," *Mind*, vol. x.

of simultaneous light- and colour-contrast may be explained by the principle that, of excitation-processes due to stimuli simultaneously applied to different parts of the retina, the more intense process tends to weaken the less intense, and the more so the nearer together in the retina are the two groups of nerve-endings stimulated, and the more closely allied in quality are the two sensations.

The principle of competition is here readily applicable, and the physiological mechanism by which the drainage of the one retino-cerebral tract by the other may be brought about may be conceived to be very similar to that sketched above (p. 175) as the most probable form in the case of the reciprocal inhibitions of antagonistic muscle-groups. We may regard the retino-cerebral tract in simple schematic form as consisting of chains of neurones lying side by side, each one leading from a retinal element through the visual cortex to some motor element; and we may conceive the activity of the tract as consisting in the flow of energy or neurin, liberated in it under the influence of stimulation, towards the motor neurones, the visual sensation being determined by the discharge of neurin across the synapses of the cortex which unite successive neurones of each chain. The intensity of the sensation determined by the activity of any one such chain must be in some way a function of the quantity of neurin transmitted by the cortical synapses in unit of time.

If now we suppose that any two such chains of neurones lying side by side are united in their afferent parts by collateral fibres in the way suggested above (p. 175), we shall see that so long as two such chains—*a* and *b*—are equally intensely excited these cross-connections will not be called into action; but if one of two such chains, *a*, be more intensely excited than the other, *b*, it becomes a path of lower resistance, and therefore attracts to itself a part of the energy liberated in the chain *b*. When chain *a* is more intensely stimulated, the cortical synapses of chain *b* will therefore transmit less energy per unit of time than when both are equally stimulated, and the intensity of the sensation determined by the stimulus to chain *b* will be

diminished. On the other hand, chain *a* will transmit through its cortical synapses a larger quantity of neurin per unit of time than it would transmit when chain *b* is equally intensely stimulated or is not at all stimulated. If, then, experiments can be devised to show unmistakably that of two unequally intense sensations excited by unequally intense stimuli applied to neighbouring retinal elements, the more intense sensation gains in intensity through the presence of the other, while the less intense loses in intensity, we may fairly suppose that the gain of the one is at the expense of the other; and such experiments will go far to justify the conception that inhibition is one result of competition between simultaneously excited nerve-tracts, of the draining of the free energy of the one tract into the other tract.

Such experiments I have been able to devise, but before describing them I refer to an observation, already published in my papers on visual sensations,[1] which seems to me especially significant in this connexion. Two patches of white light, a brighter one *a* and a less bright one *b*, are arranged upon a dark ground at such a distance apart as subtends an angle of about 30° at the eye, and a point midway between them is steadily fixated. If the two patches are simultaneously exposed to the eye, or if the duller one *b* is first exposed and the brighter one *a* a few seconds later, *b* merely exhibits some distinct loss of brightness at the moment of exposure of *a*; but if the rays from *b* are allowed to fall upon one part of the retina for some fifteen or more seconds before *a* is exposed, then on sudden exposure of *a*, *b* disappears suddenly and completely from consciousness. If then the fixation is maintained, *b* returns to consciousness after some seconds, at first feebly, but gradually increases in intensity. It seems that the activity of the tract excited by *b* during fifteen seconds produces fatigue of that tract, a fatigue chiefly affecting in all probability the synapses of the cortex. This fatigue suffices to raise the resistance of the tract *b* to such a degree, that on the exci-

[1] *Loc. cit.*, p. 179.

tation of tract *a* the stream of energy liberated in the afferent neurones of the former tract *b* is completely diverted to the latter, until, after some ten or more seconds of activity of tract *a*, fatigue raises its resistance to such a point that the stream of neurin is no longer wholly diverted from tract *b*, and the image of *b* returns to consciousness; then as increasing fatigue still further raises the resistance of tract *a*, more and more of the energy liberated in tract *b* finds its way through the cortical synapses of that tract, and the image of *b* is gradually restored to its normal brightness.

A similar sudden and complete inhibition may occur when the two patches of light are of equal physical brightness, if the one be allowed to act on the retina for a longer period before the exposure of the other.

I regard this case of inhibition as specially important, because the part played by fatigue in determining the sudden and complete inhibition is readily explained by my scheme of the inhibitory mechanism, while it is not easily to be explained in terms of any of the other hypotheses that were reviewed above.

I conclude this paper with the description of some experiments that seem to me to prove that the inhibiting neural process gains in intensity at the expense of the process which it inhibits and in proportion to the amount of inhibition that it produces.

It is commonly asserted that a patch of white upon a darker ground appears brighter than a similar patch upon a ground equally bright with itself. I should be glad to accept this statement as evidence in favour of my hypothesis if it were clear that the sensation is really increased in brightness in the same sense that a gray patch loses in brightness through being put upon a white ground. But I have not been able to satisfy myself that the apparent brightening in the former case is due to anything more than psychological contrast (in the sense of Helmholtz), which certainly plays a considerable part in such cases and is a far more complex process than physiological contrast.

If an accurately-centred white disc be put upon a larger

black disc and whirled on the colour-wheel, there is no brightening of the border of the white disc perceptible with certainty if successive contrast be carefully avoided. But if the ground surrounding the white disc be made brighter by introducing a white sector into the black disc, then on rapid rotation the border of the white disc does show some brightening; and this is most marked when the brightness of the ground is but little inferior to that of the white disc itself.[1] But here again the effect is so slight that I would attach no importance to it, were it not that the indication it affords is strongly supported by the following observations:—

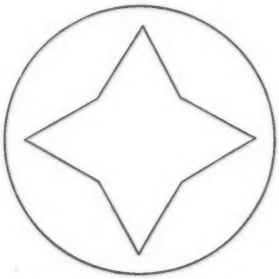

FIG. 2.

A star of stout white paper of the form shown in fig. 2 is pasted upon a large disc of black cardboard (really, of course, a dark gray surface), and this is whirled on a colour-top at a speed sufficient to give complete fusion of the alternating black and white surfaces. There then appears a white disc surrounded by a broad zone of gray, of which the brightness diminishes gradually and smoothly towards the periphery.

[1] This effect is more strongly marked when both areas are made up of black and white sectors, and when the rate of rotation is insufficient to give complete fusion and both areas exhibit a coarse flicker. The area containing a slightly larger proportion of white then shows a very marked brightening where it adjoins the area with a smaller proportion of white, and the latter shows a very marked darkening in the neighbourhood of the line of junction.

But the transition from the white centre to the dark ground is not a simple diminution of brightness. The white centre is separated from the broad gray zone by a band of greater brightness, and the gray zone is separated from the dark gray ground by a band of blackness. The former, the bright band, is much the more obvious and striking of the two; it lies upon the periphery of the white disc, which constitutes the centre of the star, and its subjective character is proved by the fact that its width subtends an approximately constant angle at the eye as the distance between the disc and the eye is increased or diminished, while, of course, the objective features of the disc subtend a smaller angle the greater the distance of the eye. As the eye is carried further from the disc the white band, therefore, spreads inwards towards the centre, appearing to increase in width. The dark band, separating the gray zone from the darker gray ground, is of about the same width as the bright band, and it

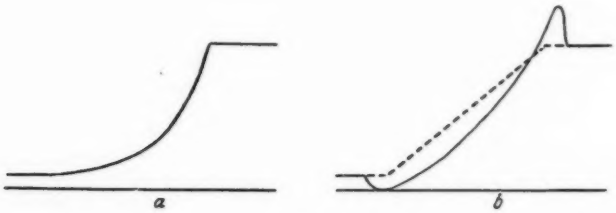

FIG. 3.

also preserves its absolute width as the position of the eye is changed. We may roughly represent the stimulus-values along any radial line of the disc by the curve, fig. 3*a*. Then, in the absence of any subjective modification of the sensation-values by interaction of the processes in neighbouring retino-cerebral elements, the curve of sensation-values along the same line would be represented approximately by the dotted line in fig. 3*b*. But in place of that we find that the sensation-values must be represented by a curve of the form of the continued line in fig. 3*b*.

That is to say, the sensation-intensity is subjectively diminished about the peripheral border of the gray zone and increased about its central border; the brightness of the whole slope of sensation is heaped up about the top of the slope. The exact form of the curve representing the slope of sensation is a matter of minor importance in the present connection.[1] The feature of special interest is the heaping up of the brightness at the top of the slope of sensation. The increase of brightness is very marked, and under favourable conditions[2] the bright band exceeds the whole centre in brightness by an amount fully equal to the difference in brightness between a disc of white paper and one made up of 280° of white paper and 80° of black cloth. The dependence of the brightness of the band upon the presence of the gray zone of diminishing brightness is proved by the following experiment:—Two similar discs (each made up of a white star on a dark ground, like that in fig. 2), are rotated side by side. On either one appears a bright band just within the edge of the white centre of the star, and the two bands are equally bright. The arms of the one star are then shortened by successive circular cuts concentric with the disc. On rotation, after each such shortening of the arms of one star, the bright band upon that disc appears less bright than at the preceding stage, until, when the arms of the star are less than 5 mm. in length, it can no longer be perceived.[3]

An interesting variety of the experiment may be made by cutting the star in form shown in fig. 4. On rotation

[1] Prof. Lloyd Morgan (Croonian Lecture, *Proc. Roy. Soc.*, vol. lxviii., 1901), has described how the curve of increasing brightness, given by ten equal steps of sensation-value, is modified when the ten steps are replaced by a band of continuously graded stimulus-value, the position of the sensation midway in brightness between black and white being altered relatively to the ends of the series. This effect is probably related to the phenomenon described above, but since Prof. Lloyd Morgan makes no mention of this bright band, I do not think that it would be profitable at present to discuss the relation between the two effects.

[2] The degree of increase of brightness depends upon the form of the curve of stimulus-value and upon the width of the angle subtended at the eye by the gray zone, but the exact determination of the most favourable conditions presents great difficulties.

[3] It will be noticed that this experiment bars out any attempt to explain the phenomenon on the lines of Hering's theory of contrast.

there appears a well-marked bright band about the region marked *b* in the figure and a less-marked bright band about the region marked *c*. The band at *b* is very distinctly brighter than any part of the zone between *b* and *c*,

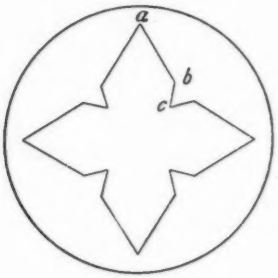

FIG. 4.

although in all this zone the intensity of the stimulus is greater than at *b*. The curve of stimulus-values may be represented by the curve of fig 5*a*, while the curve of sensation-values would (in the absence of modification by interaction of the neighbouring retino-cerebral tracts)

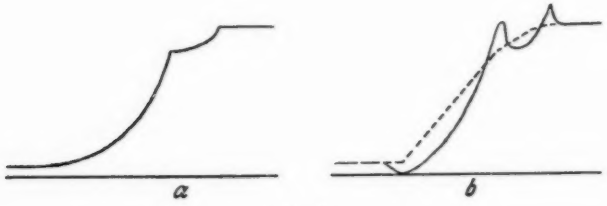

FIG. 5.

assume approximately the form of the dotted line in fig. 5*b*, whereas it actually takes the form of the continued line in fig. 5*b*. The sensation brightness is heaped up at the top of the steeper part of the slope of sensation, and to a less

degree at the top of the less steep part of the slope. In a similar way, by cutting the star so that the slope of sensation abruptly alters its angle at several points, a corresponding number of bright bands may be produced, each one being due to a heaping up of brightness at the top of a section of the slope which is succeeded by a less steep section.

We must undoubtedly seek a physiological explanation of this phenomenon, for we must believe that the brightness of the bright band is due to a greater intensity of the excitation-processes in the corresponding retino-cerebral elements. If we consider the retino-cerebral elements stimulated by the light from any one radial line of the disc, it appears that the excitation-process in the most-intensely stimulated element gains in some way at the expense of all the less-intensely stimulated elements. We can only explain this by supposing that each element in the series drains from its less-intensely excited peripheral neighbour a certain quantity of energy, and in turn is drained of a larger quantity by its more-intensely excited central neighbour. In this way there is effected a drainage of energy into the most-intensely excited elements from all the rest, with increase of intensity of the sensation determined by the former, and loss of intensity, or partial inhibition of the brightness, of the sensations determined by the latter. The mechanism of this process of drainage or competition may be such as I have suggested above (p. 175), *i.e.*, each retino-cerebral tract has its resistance lowered by stimulation of its retinal end to a degree that is proportional to the intensity of the stimulus; the more-intensely stimulated tract becomes, therefore, a path of lower resistance than its less-intensely stimulated neighbour, and, therefore, it deflects to itself and transmits through the cortical synapses some part of the stream of energy of that neighbour.

One other series of visual phenomena lends strong support to my hypothesis that inhibition is due essentially to competition for living energy or neurin. The experiments are a modification of that well-known one which goes

by the name of "Fechner's paradoxical experiment." In its simplest form this experiment may be carried out with the aid of a simple, darkly-smoked glass and any uniform white surface. If a point on the white surface is fixated by one eye (the other eye being closed), and then the other eye is opened while the dark glass is held before it, so that it looks through the glass at the same point of the white surface, the surface appears to be suddenly darkened at the moment that the second eye is opened. In a short paper, read before the Psychological Society in January, 1902, I suggested the view of the nature of inhibitory processes that is set out at greater length in this paper; and I showed that the acceptance of it would enable us to give a satisfactory explanation of this curious phenomenon, which hitherto has remained unexplained, and which seems likely to remain inexplicable for any of the current theories of inhibition.

Without repeating the exposition of my theory of the phenomenon, I wish to describe here only one of the series of experiments on which it is founded. Two plates of milk-glass are set vertically side by side in a wooden framework.[1] On each appears a circular patch of transmitted white light, the rest of the field of view being dark. The centres of the two circles are separated by a distance equal to my interocular distance. The circle on the left side, L, is of constant brightness and 25 mm. in diameter. The circle on the right side, R, is bounded by an iris diaphragm so that its diameter can be varied from 2 mm. to 40 mm., and by a simple arrangement the brightness of the light transmitted by it can be varied simultaneously[2] in all its parts from zero to about one-fourth of the objective brightness of the light transmitted through the other circle. A vertical black screen is set up midway between the two circles and the forehead rests against the edge of this at a distance of about 25 cm. from the circles. If then the eyes be brought

[1] The portable dart cabinet described in *Mind*, vol. x.

[2] This arrangement consists in using as the source of illumination of the disc, light transmitted through a narrow strip of a second plate of milk glass placed between the former and the light, at a distance of about 3 cm. from the former plate and parallel to it.

into the primary position, the images of the centres of the two circles, L and R, fall upon corresponding areas of the two retinæ, and therefore give rise to a single image in consciousness. If now R be made equal in diameter to L, and, while the eyes remain in the primary position and the image of L falling upon the centre of the left retina, the brightness of R be suddenly raised from zero to a degree of brightness about one-tenth that of L, the brightness of the image of the circle diminishes considerably at that moment, and continues so diminished for some few seconds, after which a periodic brightening and darkening is apt to set in, owing to rivalry of the two fields. This experiment, which I will name *a*, gives the Fechner-effect, pure and simple.

If now this procedure is repeated and at each repetition the diameter of the circle R is diminished by 2 or 3 mm., the following effects constantly appear :—

(*b*) When R is 20 mm. in diameter, darkening of the combined areas of the two circles occurs on exposure of R, but is less marked than in *a*.

(*c*) When R is 15 mm. no darkening of the combined image occurs ; the central part, consisting of the combined image, is not at all, or but very slightly, changed, while the peripheral zone of L, surrounding the combined image, is markedly darkened.

(*d*) When R is 10 mm. the central area of L on which R falls is brightened at the moment of exposure of R, while the peripheral part of L, especially the zone immediately surrounding the brightened centre, is markedly darkened.

(*e*) All diameters of R from 10 to 2 mm. give effects similar to (*d*).

If, now, starting again from equality of the circles, R be made larger at each step, similar, though less marked, effects are produced.

(*f*) When R is 30 mm., L darkens as R is illuminated, but less markedly than when R is only equal in diameter to L.

(*g*) When R is 35 mm., there occurs no perceptible change of brightness in L, while the peripheral parts of R are almost invisible.

(*h*) When R is 40 mm., L brightens distinctly on exposure of R, while the peripheral parts of R are hardly perceptible.

The experiment may be varied in many ways, and the effects are always similar to those described above. Thus, while R is equal in diameter to L, a sheet of blackened perforated zinc with holes 4 mm. in diameter may be put over R, so that on illumination it appears as a group of dull white circlets on a dark ground. Then if, as before, L is fixated and R illuminated (to one-tenth brightness of L), there is no Fechner-effect, no darkening, but a brightening of the parts of L with which the circlets of R coincide, while the rest of L is darkened to extinction, so that the combined field appears as bright white circlets on a dark ground. If the perforated zinc be put over L in place of R the effect is similar—a brightening of the combined areas, and an extinction of the intervening areas of R.

It appears, then, that in all cases in which a binocularly combined area of the darker and brighter fields is surrounded by an area of monocular brightness, the Fechner effect does not appear, *i.e.*, the appearance of the darker field does not darken the brighter field, but brightens it, while the surrounding monocular area loses markedly in brightness; and the brightening of the combined field is roughly proportional to the extent of the darkened monocular area and to the degree of its loss of brightness.[1] It would appear, in fact, that the loss of brightness of the monocular field is a necessary condition of the gain of brightness of the combined field, or that, in other words, the inhibiting process gains in intensity at the expense of the inhibited process. There is no need to discuss in detail the mechanism of these effects, and it is obvious that these phenomena, like those of the

[1] It is clear that whatever be the exact nature of the connections between the central parts of the retino-cerebral tracts of the two eyes, the excitation-processes in the central elements connected with corresponding points in the two retinæ reciprocally support or reinforce one another, so that in the general competition between the parts of the retino-cerebral tract of one eye, those thus reinforced or supported are enabled to drain the parts not so reinforced, and so to gain at their expense. This principle seems to give the true explanation of all those cases of inhibition which have been classed together as instances of the predominance of contours in the struggle of dissimilar visual fields.

experiments with the discs described above, afford a good experimental basis for the hypothesis that inhibitory effects in the nervous system result from a process of competition between the inhibiting and the inhibited tract, the former draining to itself a part of the energy liberated in the latter. They show, at least, that the hypothesis is capable of performing the true function of an hypothesis, namely, the affording of guidance in experiment and observation.

ON "STRINGHALT" AND "SHIVERING" IN HORSES. A STUDY IN COMPARATIVE NEURO-PATHOLOGY.

BY CHALMERS WATSON, M.B., F.R.C.P.E.

In a paper on the "Pathogenesis of Tabes and Allied Diseases in the Cord," published in the *British Medical Journal*, June 1, 1901, I drew attention to the value of a study of the clinical and pathological features of disease of the nervous system in lower animals as a means of advancing our knowledge of allied affections in man. The object of this communication is to draw further attention to this field of research. The tissues of lower animals exhibiting evidence of nervous disease can be obtained at an early stage of the morbid process when the pathological picture is as yet unobscured by the secondary and non-essential changes which so frequently complicate the picture in the human subject. As a result of my earlier investigations on cases of disease of the nervous system in animals, I found that the *post-mortem* appearances present in the central nervous system bear a close parallel to the appearances presented by certain affections in the human subject. These earlier investigations bore especially on lesions in the posterior columns and postero-lateral regions of the cord. The present one involves the consideration of lesions of (*a*) the peripheral nerves, and (*b*) the different columns of the cord indiscriminately. The results of this investigation throw some light on the causation of two important diseases of horses—Stringhalt and Shivering—the morbid anatomy and histology of which, so far as the writer knows, have not previously been ascertained. In a former paper I suggested that the clinical symptoms of a well-marked case

of "shivering" indicated that the nature and distributions of the lesions would be similar to those encountered in disseminated sclerosis in the human subject.

Clinical Record.

The subject was a light-legged, well-bred horse, 15½ hands, aged 15 years or more, that had been able for work up to three months before the date at which he came under observation. His work was light, that of going in a fishmonger's cart in a level country. His owner informed me that the animal had occasionally been off work for a few days or so from "lameness." When the animal first came under my notice it had just completed a ten mile walk, and the "nervous" symptoms were extremely pronounced. The spastic movements of the hind limbs characteristic of Stringhalt were very striking; closer investigation revealed the existence of marked paresis or paralysis of the hind limbs with a drooping of the hind quarters and distinct muscular atrophy of a widespread character. The tail was elevated and quivered on movement. A degree of inco-ordination was present, being indicated by the manner in which the hind limbs were brought to the ground. When an attempt was made to back him, the movements set up some muscular tremors of the muscles of the tail and hind quarters (shivering). Through the kindness of Principal Williams, F.R.C.V.S., the animal was housed for a few days at the New Veterinary College, Edinburgh, and a cinematograph was taken of the movements. A small sinus in the left groin was now discovered, this leading to a "scirrhous" cord, the result of a septic infection at castration when the animal was 4 or 5 years old. There were no eye disturbances and no defects in the bladder or rectal functions. After observation for a few days the animal was shot.

Post-Mortem Examination.

This was made immediately after death, and in conducting it I had the assistance of Mr. Henry Taylor, F.R.C.V.S., New College. The gross pathological appearances found were as follows:—

Thorax.—(*a*) Marked hypertrophy of the wall of left ventricle, the muscle of which was firm and apparently healthy.

(*b*) The lungs showed a moderate degree of emphysema, and also chronic venous congestion at the bases.

(*c*) *Abdomen.*—The *spleen* was of normal size; the trabeculæ appeared unduly prominent; it was otherwise normal.

(*d*) The *liver* showed slight congestion and doubtful fatty change; in other respects it presented a remarkably healthy appearance.

(*e*) The *kidneys.*—The *right* kidney was of normal size and appearance except for some congestion of vessels and thickening of the large arteries.

The *left* kidney was very much enlarged, being almost twice the size of its fellow; the cortex and medulla were markedly pale and were softer in consistence than normal. The marked pallor of the cortex was accentuated by the presence of white lines radiating out from the papillary layer to the surface. There was no appearance of abscess formations or of focal necrosis. The walls of the larger arteries did not show the same degree of thickening as was present in the right kidney. The left ureter and also the bladder were markedly thickened.

(*f*) The *gastro-intestinal tract* showed numerous patches of acute and chronic congestion.

(*g*) The "*scirrhous*" *cord* presented the form of a very large tumour, estimated to weigh between eight and ten pounds. It was removed from the region of the left spermatic cord. The tumour was composed of fibrous tissue enclosing many abscesses, varying in size from a bean to a small orange. A bacteriological report on the pus is given below.

The *Bone Marrow* was unusually red in appearance, and in the long bones there were areas in which the marrow had undergone a gelatinous degeneration.

Microscopic Examination.

This may conveniently be considered in the following order:—(*a*) The vessels in the central and peripheral nervous system; (*b*) the vessels of other systems and viscera; (*c*) the changes in the cells and fibres of the nervous system; (*d*) the renal changes; (*e*) bacteriological report.

(*a*) *The Vessels in the Central and Peripheral Nervous System.*

The vessels in the nervous system showed a considerable degree of hyaline and fibroid thickening. The arteries and capillaries in the brain and cord were much less seriously affected than those of the peripheral nerves. To take the last mentioned

first, we have a good illustration in fig. 1, a longitudinal section of a piece of the popliteal nerve, which shows four arterioles with considerably thickened walls. A high power view of one of these vessels shows a marked cellular proliferation in the vessel wall (fig. 2). An even more striking picture is seen in fig. 3, a longitudinal section of a piece of sciatic nerve, which shows that the normal appearance has been lost, owing to the dense formation of fully formed fibrous tissues and a cellular overgrowth of the connective tissues of the vessel sheaths and neurilemma. A further indication of the widespread nature of this vascular change is given in fig. 4, taken from the artery supplying the phrenic nerve; the figure shows hypertrophy both of the middle and outer coats. As illustrations of the type and extent of the disease in the cord, the reader is referred to figs. 5 and 6; fig. 5 shows to the right a vessel in a state of marked hyaline thickening; this vessel was probably impervious. The vessels to the left show distinct fibroid thickening. A similar hyaline thickening of the walls of one of the pre-capillaries is seen in fig. 6, this vessel being, however, abnormally dilated. The capillaries in the brain show marked cellular hyperplasia of their walls. Fig. 7 shows an empty cerebral capillary, the walls of which are considerably thickened. Some idea of the severity of this change may be learned from a study of a normal brain capillary stained by the same process. This is seen in fig. 8, in which the walls of a healthy capillary are practically invisible, and are only represented by three or at most four nuclei.

(*b*) *Other Vessels.*

The other changes present are mainly affections of a nature similar to those already described. In the case of the heart as in the kidney the thickening of the vessels involved the inner coat to a greater extent than the middle coat; while in the other systemic arteries examined the hypertrophy was mainly confined to the tunica media. In fig. 9 we have a good illustration of the extent of the vascular disease in the coronary arteries; a methyl violet process of staining shows an enormous thickening of the inner coat. Fig. 10 shows a localised warty thickening of the inner coat of a large branch of the posterior tibial artery, this thickening being of a cellular nature, and traversed at different levels by the separated fibres of the internal elastic lamina. Fig. 11 is a low power view of the same vessel as fig. 10, and shows marked hypertrophy of middle coat of the vessel with

extensive formation of new capillaries throughout. The normal internal elastic lamina is well seen at the upper part of the plate. A similar hypertrophy of the middle coat is seen in fig. 12, taken from the artery accompanying the posterior tibial nerve, which shows the curious manner in which the inner coat accommodated itself to the change in the middle coat. While the inner coat was comparatively slightly involved in the case of the general systemic vessels, the outer coat did not escape; this is shown by fig. 13, taken from a cutaneous artery from a part of skin that was not exposed to intermittent pressure by harness. The smaller vessels in the alimentary canal were also affected. Fig. 14 shows three small arterioles from the submucous coat of the ileum, where the lumen of some vessels was obliterated and in other cases seriously diminished by marked fibrous thickening. The muscular fibres in the trabeculæ of the spleen were in considerable excess of the normal. The presence of emphysema of the lungs (see p. 193) indicates a previous obliteration of many pulmonary capillaries. This change was accompanied by a considerable degree of chronic venous congestion and sclerosis of the walls of the air vesicles. The latter is depicted in fig. 22.

(c) *The Changes in the Nerve Elements.*

Having considered the changes present in the nutrient vessels in the nervous system, we pass now to the consideration of the changes observed in the nerve elements themselves. A moderate degree of degeneration of the nerve cells was present in the gray matter of the cord and in the cerebral cortex, being comparatively slightly marked in the latter situation. Fig. 15 is an example of this degeneration, which is shown by the vacuolation at the periphery of the cell and by the disappearance of the Nissl-bodies. Another illustration is seen in fig. 16, which shows varying degrees of degenerated cells in the anterior cornua in the lumbar region. The most marked degenerative changes in the nervous system were present in the peripheral nerves of the hind limbs.

Fig. 17 shows a longitudinal section of a piece of the popliteal nerve stained by the Weigert-Pal method; this should be compared with fig. 18, a similarly stained preparation from a healthy nerve of the fore limb. The sciatic and posterior tibial nerves also showed marked degeneration of nerve fibres (Weigert-Pal). A few degenerated fibres were also found in the phrenic nerves. The Weigert-Pal method of staining showed no degeneration in the cord, but the Marchi process showed diffuse early degenera-

tion of the nerve fibres in all parts of the cord, more especially in the posterior columns in the lumbar regions. Figs. 19 and 20 are a low and high power view of the posterior columns in the lumbar regions, showing different stages of degeneration. An almost equal degree of degeneration was present in the anterior columns of the cord in the cervical region. Fig. 21 from the cervical cord shows degenerated efferent fibres which were pro-probably associated with the degenerated cells seen in figs. 16 and 17.

(*d*) *Renal Changes.*[1]

On Microscopic Examination, the *right kidney*, which to the unaided eye showed only marked thickening of the walls of its larger vessels, showed the following conditions:—

(*a*) A number of hyaline casts in the tubules.

(*b*) A moderate degree of cellular proliferation in and around some glomeruli.

(*c*) Several foci of numerous small round cells like a miliary abscess. The organ was otherwise normal, with the exception of the marked vascular disease already described, which is depicted in figs. 23-24.

Fig. 23 shows a large artery with diminished lumen and thickened walls, also very marked over growths of connective tissue of the adventitia and peri-adventitial tissue with hyaline degeneration of new capillaries.

Fig. 24 shows the same vessel as in fig. 23, and indicates that the thickening involves partly the tunica intima and partly the tunica media.

The *left kidney*.—Reference was previously made to the pronounced nature of the gross changes present in this organ; which did not, however, show the same degree of arterial disease as its fellow. The histological changes were very striking throughout, mainly consisting in a very great invasion of small round cells, with considerable fibrous changes, and chronic catarrh of the tubules. The increase in size of the organ to double the normal appeared to be mainly due to the presence of leucocytes, most of which were of the polymorpho-nucleated variety. The tubules were in parts atrophied and in parts dilated. Some glomeruli showed marked thickening of their walls.

[1] As the renal changes are of considerable interest from the point of view of a chronic general infection of the nervous and other systems, they will be more fully described and illustrated in a later communication.

(e) *Bacteriological Report.*

A bacteriological report on the pus from the "Scirrhous cord" was kindly given me by Dr. Shennan, who reported as follows: "Plate cultures separated out what appeared to be different colonies; microscopically three of them seemed distinctive, and I tried them in guinea pigs, but without result. I also tried them after anaërobic cultivation, with the same result. Two of them were short rods with end staining; the other was a diplococcus. They were all evidently non-pathogenic. None of the ordinary pyogenic organisms were present."

Remarks on the Case.

Two aspects of the case call for further comment.

(1) The light thrown on the pathology of certain common diseases of the horse, and (2) Suggestive lessons for the student of pathological processes in the human subject.

"Stringhalt" and "Shivering" in horses.—These diseases frequently coexist in the same subject, but cases of Stringhalt alone are very common. So far as I have been able to ascertain, the morbid anatomy and histology of these diseases have not previously been elucidated, and the present record is the first serious attempt at their investigation. I am well aware that the term Stringhalt is somewhat indiscriminately applied by some veterinarians to certain types of movement in horses which are the subject of some local disease in the limb, *e.g.*, at the hock joint. In this paper the use of the term has been restricted to the more commonly recognised form of true stringhalt, viz., cases in which the "nervous" symptoms and physical signs are alone present. The present record shows that the stringhalt was the result of a chronic interstitial neuritis with secondary degeneration of the nerve fibres in the main nerves of the hind limbs. The symptoms of "shivering" are adequately explained by the existence of the degeneration in the cells and fibres of the spinal cord in combination with the peripheral nerve lesions described; in

other cases they might be adequately accounted for by the presence of a more advanced cord lesion alone. I do not maintain that all cases of stringhalt are to be thus explained. In a former paper I have shown that typical symptoms of this disease may arise as a result of a lesion in the motor part of the central nervous system within the brain, and I have reason for believing that they may be dependent on a lesion mainly confined to the lateral columns of the cord. This is only what we should expect.

The paresis of the hind limbs and manifest muscular atrophy in this subject are adequately explained by the existence of the lesions in the cord and peripheral nerves alone; and if we add to that the profound state of chronic acute intoxication to which this animal was habitually exposed from the condition of the kidneys and spermatic cord, and to a lesser extent from the state of the mucous membrane lining the respiratory and alimentary tracts, we have reason for marvelling at the manner in which the animal had lived and worked for so many years. The facts are to my mind only explicable on the assumption that the animal had long since acquired a considerable degree of immunity to the influence of the bacteria whose action had produced the remarkable condition of the spermatic cord and kidney—a view which gains some support from the bacteriological report kindly given by Dr. Shennan.

Before passing to a consideration of the second point of these remarks, attention may be directed to the interest of the case from the standpoint of comparative renal pathology.

Lessons Suggested to the Students of Allied Pathological Processes in the Human Subject.

When we have regard to the widespread hypertrophy of the middle and to a lesser extent the outer coat of the main arteries, the pronounced thickening of the tunica intima present in the vessels of the heart and kidney, the hyaline and fibroid degeneration of vessels in the cord, the fibrosis of capillaries in the brain, and, most striking of all,

the advanced arterial and capillary disease in the peripheral nerves, we have a typical object-lesson of general vascular disease. The full considerations of any one of these points is beyond the scope of the present paper. It must suffice to again draw attention to the points here summarised, which suggest that the further study of similar cases in comparative pathology would materially enhance our knowledge of some keenly-debated problems in vascular disease in the human subject. With regard to the relationship of the vascular disease to the degeneration of the nerve-elements in this case, there can be no question as to which is to be regarded as the primary affection. A careful investigation of this point showed conclusively that the disease of the vessels was considerably in excess of the degeneration of the nervous structures. It was indeed matter for comment that the degeneration of the peripheral nerve-fibres was not more severe. The results of this investigation are thus in harmony with my conclusions from previous researches in comparative neuro-pathology in clearly indicating the primary importance of vascular lesions in the production of some chronic nervous diseases in the lower animals, and, as previously pointed out, this field of investigation is one which presents a more likely prospect of advancing our knowledge than is offered by observations on cases of disease in the human subject.

The results of this investigation throw no light on the important question of the nature of the circulating irritant responsible for the production of this widespread vascular disease and resulting degeneration of nervous structures. The record does indeed suggest that the primary source of infection was the scirrhous cord, but as this is a comparatively rare disease, and as stringhalt and shivering are not rare diseases, it is obvious that there must be other sources of infection, and of these the most frequent, in my opinion, is the alimentary tract.

In concluding, a word may be said on the subject of immunity to which reference was made in the paper. There is reason to believe that the arrest of not a few cases of chronic organic disease of the nervous system in the human

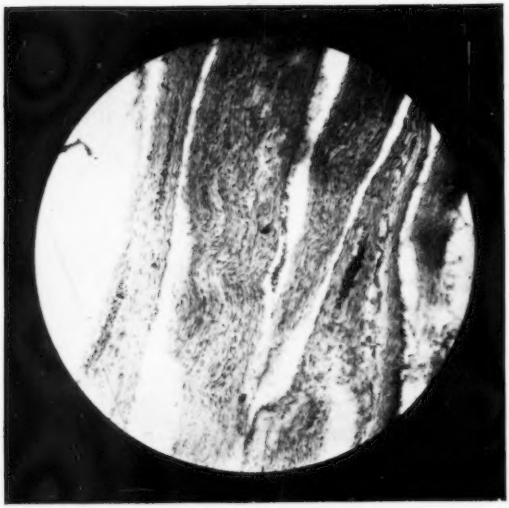

FIG. 1.—Popliteal nerve (low power). To show four vessels with thickened walls (*cf.* Fig. 2).

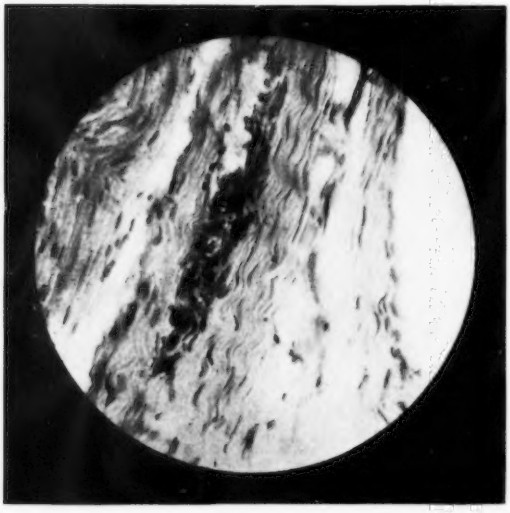

FIG. 2.—Higher magnification of one of the vessels in Fig. 1. Note the cellular proliferation of the vessel wall.

FIG. 3.—Sciatic nerve. Shows very marked interstitial changes, both old and recent. The normal outline of the nerve is indicated in the right upper part of the field.

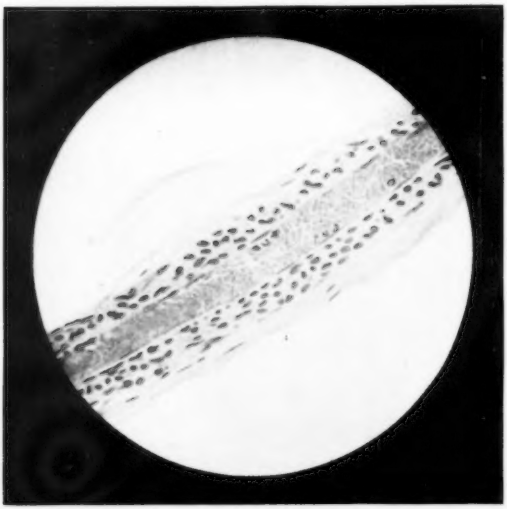

FIG. 4.—Nutrient artery of phrenic nerve, to show hypertrophy of the middle and outer coats.

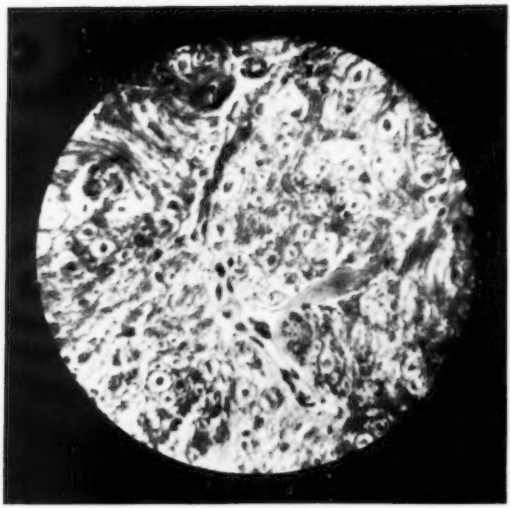

FIG. 5.—Vessels in the cord, showing hyaline and fibroid thickening. The hyaline swelling is seen in the vessel running horizontally and to the right of the field.

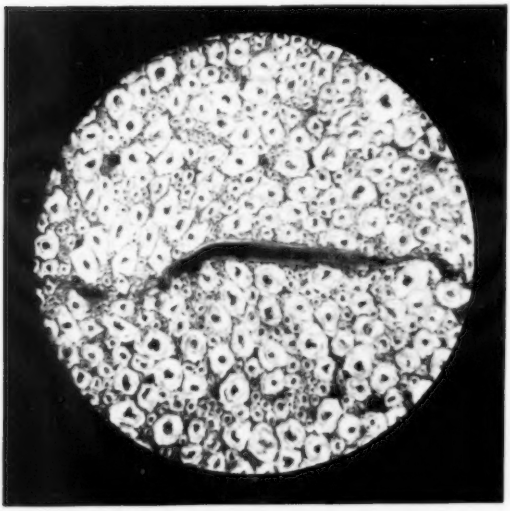

FIG. 6.—Hyaline thickening of wall of capillary in the cord.

FIG. 7.—Cerebral capillary (empty), showing marked thickening of its wall (*cf.* Fig. 8).

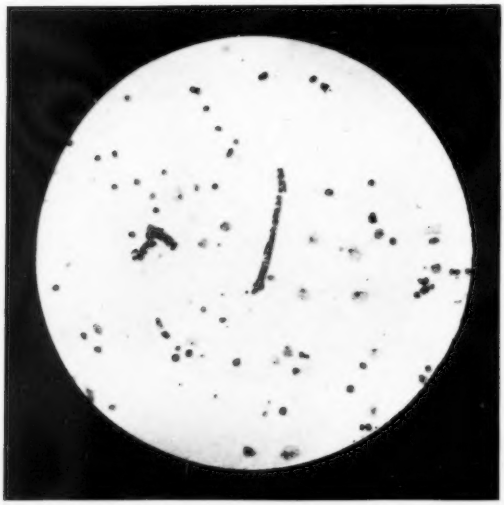

FIG. 8.—A normal capillary full of blood. Note that the walls are practically invisible.

FIG. 9.—A coronary artery, with considerably thickened walls. The dark line is the internal elastic lamina.

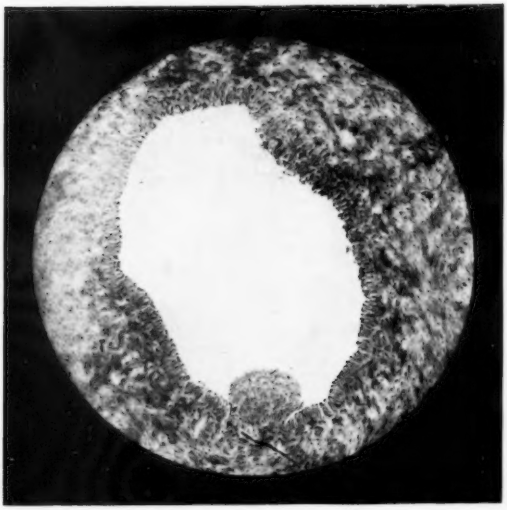

FIG. 10.—Branch of posterior tibial artery, showing warty projection of tunica intima.

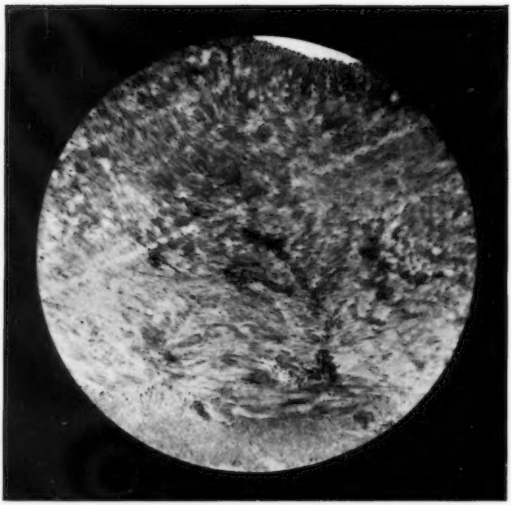

FIG. 11.—Same vessel as Fig. 10. To show the hypertrophy of middle coat with new formation of capillaries.

FIG. 12.—Nutrient artery of posterior tibial nerve. Note the thickening of the middle coat and plicated arrangement of the inner coat.

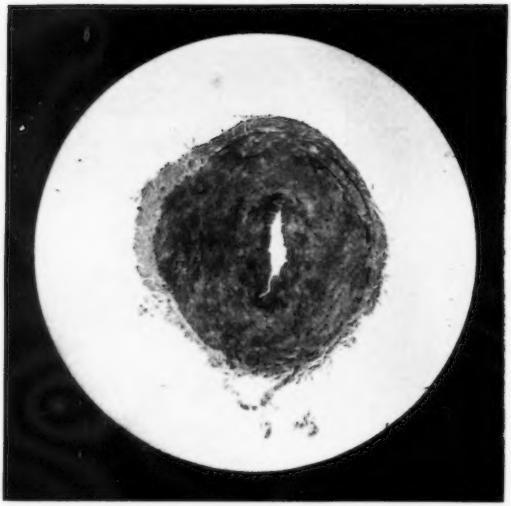

FIG. 13.—A cutaneous artery, showing hypertrophy of the middle and outer coats.

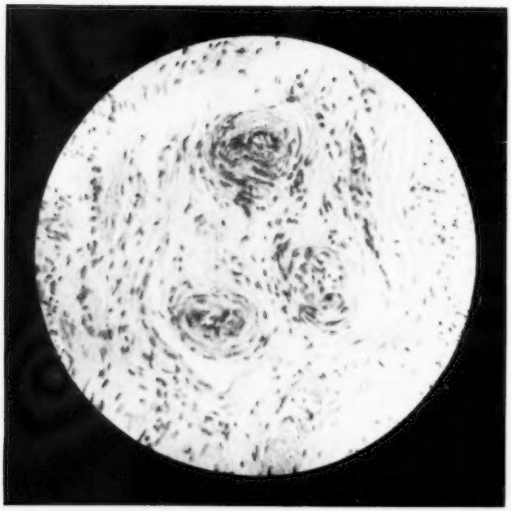

FIG. 14.—Diseased vessels in submucous coat of the intestine.

FIG. 15.—Anterior cornu. Note the vacuolation at the periphery of the cell and disappearance of Nissl bodies.

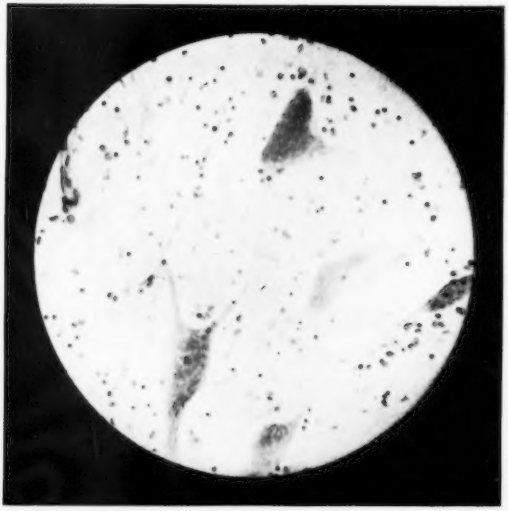

FIG. 16.—Varying stages of degeneration of nerve cells in anterior cornu.

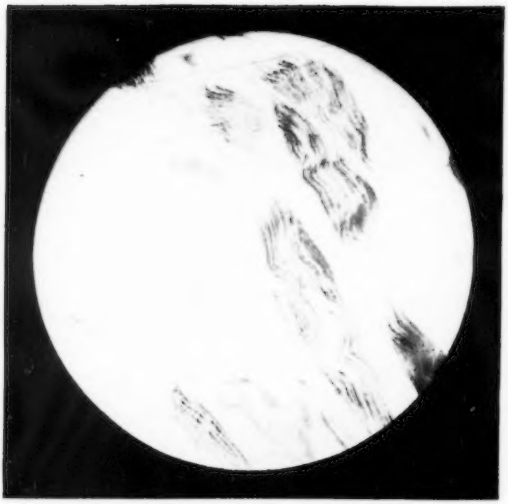

FIG. 17.—Popliteal nerve (Weigert-Pal), *cf.* Fig. 18.

FIG. 18.—Healthy nerve from axilla (Weigert-Pal).

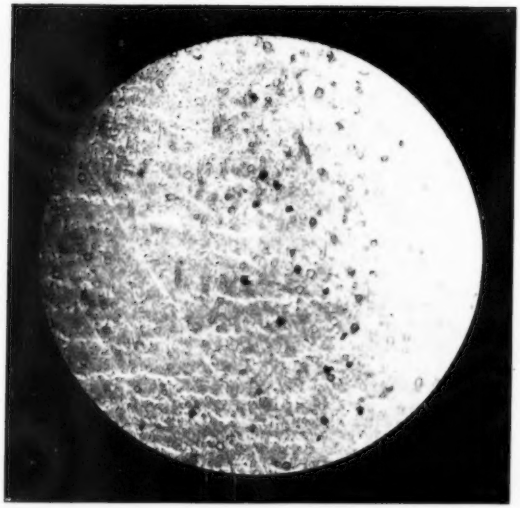

Fig. 19.—Posterior column of cord (Marchi).

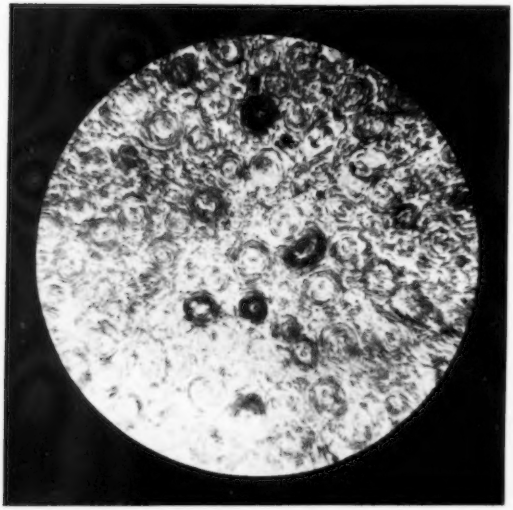

Fig. 20.—Same as Fig. 19, showing partially degenerated fibres.

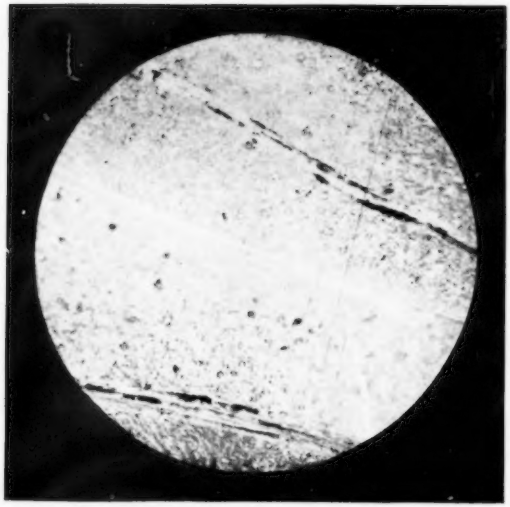

FIG. 21.—Cervical cord, showing degenerated efferent fibres (Marchi).

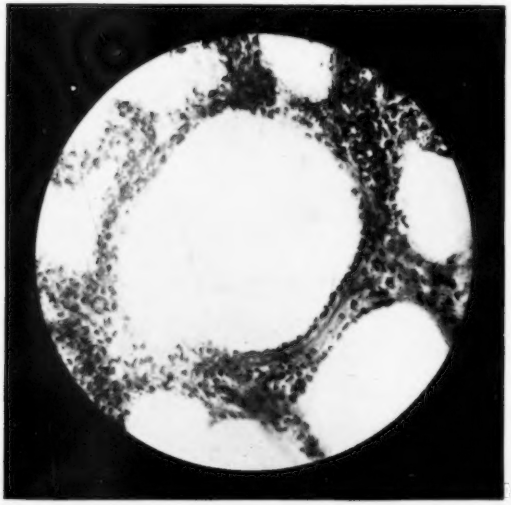

FIG. 22.—Lung, showing marked fibrous overgrowth of the walls of the air vesicles.

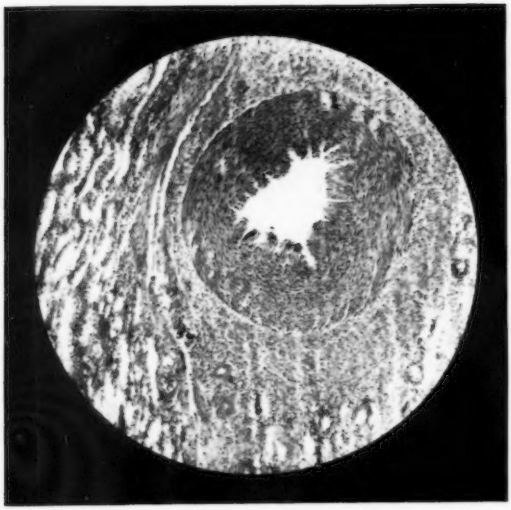

FIG. 23.—Right kidney. Shows thickening of walls of artery with peri-adventitial overgrowth and hyaline degeneration of capillaries.

FIG. 24.—Right kidney. Note the thickening of the tunica intima.

subject is due to the establishment of a degree of immunity like that present in the subject of this paper.

I have great pleasure in expressing my indebtedness to Dr. Ford Robertson, director of the Laboratory of the Scottish Asylums, for invaluable assistance in the course of this investigation; also to W. Owen Williams, President of the Royal College of Veterinary Surgeons, England, for much advice and help, and very especially to Miss Watson, L.R.C.P.E., Wellington, New Zealand, who did most of the laboratory work involved in this investigation.

A CASE OF ERB'S JUVENILE DYSTROPHY ASSOCIATED WITH BILATERAL ENLARGEMENT OF THE PAROTID AND SUBMAXILLARY GLANDS.

BY J. MICHELL CLARKE, M.A., M.D., F.R.C.P.

Physician to the Bristol General Hospital.

THE following case is remarkable for the association with fairly general and pronounced muscular atrophy of an enlargement of the parotid and, to a less degree, of the submaxillary glands. The muscular atrophy, though somewhat anomalous in type, conforms most nearly to Erb's juvenile muscular dystrophy. The swelling of the parotid appears to be a simple glandular enlargement.

The onset of both atrophy and swelling was simultaneous, without any definite cause. The swelling of the parotids has long remained stationary, and the muscular atrophy has during the last year or two apparently ceased to extend. It is difficult to see what connection there can be between the two phenomena, or whether there is any at all; possibly it is a mere coincidence in the same patient of two rare symptoms, but if so I think it is a sufficiently curious one to merit a brief record. In favour of some common cause for the parotid swelling and muscular dystrophy is, of course, the coincidence in the onset of both affections.

Frederick E., aged 18 years, complains of difficulty in walking and getting about, and of wasting of muscles.

Family history good; no other case of the same kind in it, nor of any other form of chronic paralysis. The patient is the eldest of five children, the others are healthy; I have examined them, and found no signs of muscular atrophy or hypertrophy in any.

The patient was born naturally and was quite healthy until 13 years of age. He was an active and very intelligent boy. He

FIG. 1.

FIG. 2.

FIG. 3.

had had no previous illness; the present one began insidiously, without assignable cause. The first symptom noticed was weakness of the legs, which would give way suddenly and cause him to fall down. The muscular wasting did not attract attention until he was first brought to the Bristol General Hospital at the age of 15 years; it had then the distribution described below, and from the fact that weakness began in the legs, and that when he fell he could at first pull himself up by his arms, but later became unable to do this, it is inferred that the wasting began in the lower and subsequently spread to the upper extremities. With the onset of these symptoms came simultaneously swelling of both parotid glands; this took some weeks or (?) months to develop to the present size, and then remained stationary. There was not at any time pain, redness, or tenderness in them, and no intermittent attacks of pain or increased swelling occurred. He has never had mumps. The muscular weakness progressively increased, so that he could hardly get about and could not get up again when he fell. He has never had any pain in the limbs, nor any sensory disturbance, subjective or objective. From the onset of the illness he has ceased to grow in size.

When he first came to the Hospital in 1900, he was just able to stand and to walk, his gait was somewhat tottering; he did not drag his feet. He could get out of bed, but could not get back into it again, and could not go upstairs. When placed on his back on the floor he could turn over, and get on to his hands and knees, but no further. The special senses were normal except that he was deaf; watch was heard in contact (left ear) at one inch; (right ear) he was deaf to the high notes of Galton's whistle. When admitted in 1903 the muscular wasting and general condition appeared to have practically remained stationary, and is described below. The atrophy was perhaps more marked in some of the muscles, but on the other hand he walked better, and did not fall about so much, he was now able to go up a few stairs provided the steps were low, and he was also better able to button and unbutton his clothes.

Present State.—The lad presents a remarkable appearance from the relatively large size of the head, the increased breadth of the facial aspect due to the parotid swellings, and the stunted growth of the trunk and limbs with the pronounced muscular atrophy. The head measures 22 inches in circumference, the parotid swellings give a peculiar broad appearance to the face, and the head is actually broad (14 inches between the mastoids).

The upper lip is large and long, the lower end of the nose broad, and alæ nasi wide. There is some nasal obstruction due to chronic hypertrophic rhinitis, but no adenoid growths are present.

The ears are large and prominent. There is a symmetrical enlargement of each parotid gland, to about the size of a small orange; the swellings are painless, moderately soft, elastic and lobulated. The submaxillary glands are also enlarged, but not to the same extent; the right is the larger. The parotid ducts are patent, the saliva contains KCNS and turns starch into sugar; it is normal in amount, and he does not suffer from dry mouth. *There are no enlarged lymphatic glands.* The eyeballs are somewhat prominent, the upper lids full, especially on the right side, where fulness is due to the enlarged lachrymal gland, which can be plainly felt.

The facial muscles are not wasted, the eye-movements are normal; the pupils equal and act well: the tongue, soft palate, and larynx are normal, and speech unaffected. In the face Chvostek's sign can generally be obtained.

The muscular atrophy in the trunk and limbs is very pronounced and affects in most marked degree the following muscles on both sides: deltoids, triceps, pectoralis major, sterno-mastoid, latissimus dorsi (left more than right), supinator longus (right more than left); in rather less degree, trapezius (right more than left), biceps, long flexors and extensors of wrist and fingers. In the pectorals the lower part of the right and the upper part of the left muscle are chiefly affected; the external and internal rotators of the humerus act fairly well; the supraspinati and infraspinati are not wasted and of good size, the right supraspinatus especially so.

The abductors and adductors of wrists are weak. In the hands the long extensors of the thumb are affected, especially extensor ossis metacarpi, and the thumb is adducted towards the palm; it can be opposed to the little finger. The left abductor indicis, and the hypothenar eminence are wasted. The fingers, owing to predominant action of interossei, are slightly flexed at proximal, extended at distal, phalangeal articulations.

It should be added that though the small muscles of the hands are relatively spared, yet like all the other muscles of the body they are small and poorly developed.

The serratus magnus is small, but its action is normal. The intercostal muscles are much wasted. The action of the diaphragm is normal. The erector spinæ is unaffected.

Of the abdominal muscles atrophy affects the lower two

thirds of each rectus, whilst the upper third is spared; the interdigitations between the sections of the muscle are well shown; the external oblique muscles are much atrophied, the right having almost entirely gone. There is an interval between the recti when they contract, and from the weakness of the obliques the abdominal wall above Poupart's ligament is protruded on coughing.

In the glutei there is marked wasting of the right, slight of the left. The action of the psoas-iliacus is very feeble. In the thighs the quadriceps extensor is much atrophied on each side, especially on the left, the adductors are also somewhat wasted, but the hamstring muscles appear to have escaped. Below the knee there is slight wasting of the anterior tibials and peronei, but the calf muscles have escaped. The feet are flat, with prominent internal malleoli of the ankle, and the feet cannot be dorsi-flexed beyond a right angle. The muscles show no fibrillary tremors.

On electrical stimulation the affected muscles show a diminished response, in proportion to the degree of atrophy present, to both forms of current; the most affected do not respond at all. There is no qualitative change, nor R.D.

Reflexes.—Tendon reflexes all absent. Superficial reflexes, epigastric, abdominal and cremasteric present; plantar difficult to obtain, of flexor type.[1] No alteration of sensation; no affection of special senses except that he is now totally deaf. No affection of bladder or rectum. Thoracic and abdominal organs normal.

The arrested growth of the boy has been noticed above, it affects all parts of the body, and the small size of the bones, which is, of course, most obvious in the long bones, is perhaps especially worthy of remark, as exemplifying the general arrest of growth which seems to have been coincident with the onset of muscular atrophy. The lad of 18 looks like a boy of 14 or 15. Intelligence is good.

Mr. C. H. Walker kindly took skiagrams of the long bones; nothing abnormal was observed in the process of ossification, and the skiagrams are therefore not reproduced. I am indebted to Dr. Newman Neild for the photographs.

[1] When the patient was in London, Dr. Batten, on examining him after a bath, found an extensor plantar reflex. On subsequent examination this has been noted once or twice since, but sometimes the reflex is not obtained at all.

NOTE ON "TOXIC DEGENERATION OF THE LOWER NEURONES SIMULATING PERIPHERAL NEURITIS."

BY R. T. WILLIAMSON, M.D.LOND., F.R.C.P.

Physician to the Ancoats Hospital, and Assistant Physician, Royal Infirmary, Manchester.

In an interesting article in No. 100 of BRAIN, vol. xxv., 1902, Dr. Stanley Barnes furnishes clinical and pathological evidence of a clinical type of paralysis, which resembles the paralysis seen in multiple neuritis, but which is associated with great atrophy of the hand muscles. The disease is stated to begin usually about the second or third week after a febrile affection; sensory symptoms are present, but slight in degree; the condition is said to resemble somewhat progressive muscular atrophy, but the etiology is different, and there is a "constant tendency to improvement." Dr. Barnes thinks the condition "is one of toxic degeneration of the lower neurones, the motor neurones being more particularly affected, and the pathological examination of one case which he records supports this view.

The following notes of a case which was under my care in 1899, at the Ancoats Hospital, Manchester, may be of interest in connection with this article. The case was shown at a meeting of the Manchester Medical Society in 1899, but no record of it has been published. The symptoms appear, however, to correspond to those of the clinical type which Dr. Barnes has so clearly described.

Atrophic Paralysis of the Muscles of the Hands and Forearms following an Acute Illness.

Isabella M., aged 40, was admitted as an inpatient at the Ancoats Hospital, October 10, 1899.

History.—In June, 1899, she suffered from an acute illness, in which the chief symptoms were pains all over the limbs and trunk. She was very ill for eight days, and then slowly recovered; but for three weeks she felt unwell. She returned to her work, and continued in good health until August, 1899. At that date she had a second attack of a similar nature. The symptoms commenced acutely, and consisted chiefly of pains in the limbs and back, along with a feeling of general illness and loss of appetite. There was no skin rash. The pains were most severe at the bottom of the back; in the limbs the pain was in all parts and not specially localised to the joints. (The ailment was thought to be influenza by the patient.) The acute symptoms continued for a few days, and then disappeared, but she felt very weak for several days afterwards. A week or a little longer after the pains disappeared she noticed that the hands were becoming feeble.

The right hand was first affected; she noticed difficulty in extending the little finger and ring finger; later all the fingers became affected, and gradually the whole hand became weak. Two weeks after the right hand was first affected, she noticed the commencement of similar symptoms in the left hand. The fingers of the left hand gradually dropped like those of the right; the little finger and ring finger were first affected, and then the whole hand became weak. The weakness of the arms gradually increased, but was not associated with tenderness, pain, or numbness. The legs and the bladder were not affected.

There was no history or indication of alcoholism, nor of poisoning by lead, or any other substance; also there was no history of diphtheria. The patient was almost a total abstainer from alcoholic beverages.

On admission to the hospital (October 10, 1899), the patient complained only of loss of power in both arms. There was considerable wasting of the forearm muscles and *marked atrophy of the thenar and hypothenar eminences and of the interossei muscles of each hand.* (The appearance of the hands corresponded to that shown in Plate I., second photograph in Dr. Barnes's paper.) There was "dropped" wrist on each side, and the patient was unable to extend either the fingers or the wrists. Also the grasp was very feeble on each side. She was unable to oppose the tips of the thumbs and fingers, and she was unable to separate the fingers. There was paralysis of the interossei, thenar and hypothenar muscles on each side, and of the extensor muscles of the fingers and wrists. The flexors of the fingers and wrists were

feeble, but the supinator longus and upper arm and shoulder muscles were not affected.

Electrical Examination.—The extensors of the fingers and wrist, and the thenar muscles on both sides did *not* react to a strong faradic current. The flexors of the fingers and wrist reacted to a weak faradic current, the interossei to a strong current. The reaction to galvanism was good, a weak current produced a contraction readily in the extensor muscles, and KCC was greater than ACC.

There was no pain, no tenderness, no numbness in the arms. Sensation to touch and painful impressions, and to heat and cold, was quite normal on the arms and hands. The intercostal muscles were not affected. There was no pain in the neck or back. She was able to walk quite well and there was no affection of the legs. The knee-jerks were normal, there was no ankle-clonus, no rigidity of the legs, and no abnormality in the gait. The cranial nerves were unaffected and there were no bulbar symptoms. The bladder and rectum were unaffected. The heart, the lungs, the digestive system, and the urine were normal. There was no blue lead line on the gums, no history of lead colic, and no evidence of any indication of lead poisoning. I examined the drinking water but found no evidence of the presence of lead.

The patient remained in the hospital for six weeks and improved gradually. She then attended as an outpatient and steadily regained power in the arms; but there was a little feebleness in the grasp of the hand and difficulty in buttoning the clothes, in threading a needle, and in picking up small objects, long after she was able to perform all the coarser movements of hands and arms. Also, the wasting of the small muscles of the hand could be detected for several months after leaving the hospital. In May, 1900, the motor power in the arms had completely returned; afterwards all signs of atrophy of the hand muscles disappeared. She was seen in February, 1901, in 1902, and March, 1903; there has been no return of her paralysis, and the woman remains quite well.

Remarks.

Briefly stated, the case was one of paralysis of the small muscles of the hands, and of the extensors of the fingers and wrist, with paresis of the flexors of the fingers and wrist. There was atrophy of the affected muscles, especially the

hand muscles. The paralysis followed an acute illness, the chief symptoms of which were pain in the limbs and trunk (? influenza). The legs were not affected and the knee-jerks were always present. Alcoholism and poisoning by arsenical beer could be excluded definitely, also there was no evidence of lead poisoning or diphtheria.

The diagnosis appeared to rest between acute anterior poliomyelitis at the lower cervical and first dorsal segments, and peripheral neuritis, of an unusual motor form, localised to the arms. But the symmetrical distribution of the paralysis, and the *complete* recovery, were against the diagnosis of uncomplicated acute anterior poliomyelitis in its usual form. In some respects the symptoms resembled those of a peripheral neuritis of the motor form, such as is seen in lead paralysis. But it was not possible to obtain any sign or indication of lead poisoning whatsoever; also the small muscles of the hand were markedly atrophied and paralysed at the *early* period of the disease, and the affection appeared to be directly related to the attack of acute illness (? influenza), which it immediately followed.

The foregoing account is an abstract of my notes of the case, made in 1899. At that time I recognised the peculiarities of the clinical features, but was not able to arrive at a definite diagnosis. I think there can be now little doubt that the case belongs to the clinical group, to which Dr. S. Barnes has drawn attention in his recent article, and to which he has given the name of toxic degeneration of the lower neurones.

CHOREA AND GRAVES' DISEASE.

BY G. A. SUTHERLAND, M.D., F.R.C.P.

THE nature of the association between chorea and Graves' disease is still under discussion. All are agreed that movements of a choreiform type are sometimes manifested in Graves' disease. Sir William Gowers[1] says that such movements must be regarded in some cases as a complication, although in a few cases in childhood true chorea has been supposed to exist. Professor Dieulafoy[2] says that choreiform movements may form part of the motor troubles in Graves' disease, as the tremors and the paralyses do, but that there is no true chorea associated with Graves' disease. He considers that if Graves' disease and chorea were associated maladies, and were dependent on a common cause, we should not find the sequence always the same, namely, that Graves' disease always precedes chorea and is never a sequel to it. To this last statement I wish specially to direct attention in the light of the two following cases.

Case 1.—Mrs. F., aged 26 years, was seen for the first time on January 15, 1901, when she gave the following history. During childhood she had suffered from enlarged cervical glands, and at the age of sixteen she had had an attack of rheumatic fever which confined her to bed for a month. Six months later she developed chorea, and this illness lasted for thirteen months. There had been no recurrence, but she had had a tendency to palpitation ever since. She had also noticed a trembling in the limbs for a long time, and a marked increase in general nervousness. During the past twelve months she had suffered much from palpitation and tremors, and a throbbing swelling had appeared in the neck.

[1] "Diseases of the Nervous System," vol. ii., 1893.
[2] *Clinique Medicale*, 1898, "Un cas de Maladie de Basedow."

Her father and mother are alive and healthy, but rheumatism and chorea have been common in the family of the latter. Two brothers and one sister are alive and well. One brother died of a cerebral abscess. Her younger brother has suffered from chorea. The patient has one child, a boy of two and a half years, who is healthy.

On examination the cardinal signs of Graves' disease were found to be present. She was very emotional and of an anxious temperament. There was slight exophthalmos with puffiness of both eyelids. The thyroid gland was enlarged, the left lobe more especially, and pulsating. The cardiac action was rapid, 110 to 120 per minute and somewhat heaving in character, but there was no enlargement or valvular disease of the heart. Universal fine tremors were present when the patient stood up, and were characteristic of Graves' disease. No true choreic movements were visible. Great weakness, profuse sweating, dilated pupils, and pulsating abdominal aorta were other manifestations of the same disease which were noted in this case. Under rest and tonic treatment the condition improved considerably.

Case 2.—D. R., female, was first seen at the age of 15 years. Her general health had been good except for attacks of chorea, of which she had had seven in all. The first of these had occurred at the age of 6, and others had followed in succeeding years. The last attack had come on twelve months previously, had been very persistent, and had produced weakness in the right arm and leg. Simultaneously with the onset of this last attack a swelling in the front of the neck had developed. She had never had rheumatic fever, but pains in the limbs had been frequently complained of, more especially when chorea was present. In the family history there was nothing of importance, save that an elder sister was suffering from rheumatism, mitral disease, and well marked Graves' disease.

On examination the patient was found to be a well-nourished girl, with marked choreic twitching in the right arm and leg. The thyroid gland was slightly enlarged, but there was no definite pulsation, or murmur, or thrill. The eyes had a slightly staring look, but there was no exophthalmos or delayed descent of the upper lids. There were no fine tremors. The cardiac action was rather rapid under the excitement of examination, and there was no evidence of valvular disease. The diagnosis made at the time was chorea, with slight thyroid enlargement, such as is common in girls at the age of this patient.

She was not seen again until fifteen months later (October,

1899), at the age of sixteen. During the previous July the following changes had been observed to come on rather suddenly, namely, increased swelling of the neck, tremors in all the limbs, prominence of the eyes, and rapid wasting, although she was eating well. Hot flushing and sweating, and shortness of breath on exertion had also been complained of. Definite exophthalmos was now present. The thyroid gland was enlarged, soft, and pulsating. There were typical tremors in the tongue, arms, and legs, in addition to slight choreic twitching in the limbs on the right side. There was heaving pulsation in the præcordial region, with tachycardia, and a systolic murmur at the apex, but no evidence of cardiac dilatation. It was, in short, a typical case of Graves' disease.

These two cases show that Graves' disease may follow on chorea, a sequence which Dieulafoy states has never been recorded. In the first patient the effects of the prolonged attack of chorea had never quite passed off, and she had suffered from palpitation and trembling for some time before the cardinal symptoms of Graves' disease were sufficiently marked to lead her to seek relief. In the second case the tendency to chorea was very marked, there having been seven attacks in all. She first came under observation during the transitional period between chorea and Graves' disease, and at this stage I had some difficulty in deciding as to which of these diseases she was suffering from. Choreic twitching, goitre, tachycardia, and slight staring of the eyes were the symptoms present, but as the three last were so slightly marked, and as they are not infrequently present in chorea, I finally decided on a diagnosis of chorea. The problem was still further complicated by the fact that a sister, a year older, presented the typical signs of Graves' disease. The sequence of events showed that this was really a transitional stage between chorea and Graves' disease, as from that date the choreic manifestations retired into the background, while those of Graves' disease became more and more pronounced.

Unfortunately we do not yet know the pathology of Graves' disease or of chorea, so that the names merely serve to indicate a collection of symptoms. The nervous disorders present in these diseases may not have the same

cause, but they would appear to depend on a somewhat similar affection of the central nervous system, and it may be that in childhood this produces the symptoms known as chorea, while in early adult life it is manifested by the symptoms known as Graves' disease.

When viewed clinically the two affections present many striking similarities. All are agreed that in both there is an inherited neuropathic tendency, which may have been manifested in organic nervous disease, such as insanity, or in functional nervous disorder, such as epilepsy. In many cases there is also found a history of rheumatism and heart disease amongst the immediate relatives. Statistics show that in both affections most of the sufferers are of the female sex; are possessed of a highly-strung nervous system; and are the subjects of rheumatism and heart disease. The immediate cause of an attack is frequently some sudden shock to the nervous system, it may be fright, or emotion, or disappointment. Choreic movements or general trembling may have come on directly after such shock, or may have been delayed for some weeks or months. In the last case, although symptoms characteristic of the disease are deferred, it has often been observed that the patient in the interval has not been quite herself. Mental changes have gradually developed in the form of irritability, excitability, weeping fits, or gusts of passion alternating with periods of good nature, excessive brightness, or, it may be, extreme apathy. Along with these, restlessness of the body is frequently observed, the patient being unable to settle down quietly to any occupation or even to sit still. As the disease progresses the mental changes may become more pronounced, leading to the development of dementia and delirium in chorea, and of melancholia and mania in Graves' disease. In both cases a fatal termination is often associated with acute mental symptoms. Sensory changes are conspicuous for their absence, save when hysteria is present. Paralysis and paresis of muscles are common. In chorea, paraplegia and hemiplegia are found, and in Graves' disease, paraplegia, hemiplegia, and ophthalmoplegia. In both cases a striking feature is the sudden "giving way of the legs" when the

patient is walking. The irregular spasmodic jerkings of chorea may be closely imitated by similar movements in Graves' disease, although in the latter fine tremors are more common. In both cases the movements are much increased under emotion, and cease during sleep. Thyroid enlargement is one of the characteristic features of Graves' disease, and is not uncommon in chorea. In the former affection a staring fixed look in the eye, the so-called "tragic eye," associated in pronounced cases with exophthalmos, is strongly suggestive of a similar appearance in chorea, which may also be accompanied by definite exophthalmos. Finally, as regards the treatment, Sir William Gowers'[1] remarks are interesting. Of chorea he writes, "The first important element in treatment is to secure the patient from mental and physical fatigue, and emotional excitement." Of Graves' disease he writes, "The first and most important element in treatment is to secure tranquility of mind and rest of body." Herein lies not only the alpha but also the omega of our therapeutical knowledge of the two affections.

[1] "Diseases of the Nervous System," vol. ii., 1893, pp. 621, 891.

THE FUNCTIONS OF THE FRONTAL LOBES.[1]

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THE following paper deals with the subject of the functions of the frontal lobes from the standpoint of the morbid anatomy of mental disease, and the general histology of the cerebral cortex in normal individuals and in the subjects of mental disease. That this is not one of the more common methods of studying the question constitutes the writer's excuse for adding the present contribution to an already overburdened literature.

As Monakow's admirable article (15) in the *Ergebnisse der Physiologie* contains a list of 846 publications on the subject of cerebral localisation which have appeared during the past twelve years, the writer proposes to limit himself to such references to literature as are germane to the purpose he has in view, and refers those who desire a full bibliography to the above monograph.

The present paper is especially concerned with the functions of the anterior portions of the frontal lobes, or the prefrontal region of the cerebrum. It is, however, necessary that a few preliminary remarks on general cerebral localisation and on the functions of the posterior parts of the frontal lobes should first be introduced.

Between the years 1842 and 1870, with few dissentients, it was generally accepted, chiefly owing to the writings of Flourens (8), that no localisation of function existed in the cerebrum, and even much later than the latter date Goltz (10) and also Loeb (14) have held that all regions of the cortex possess equivalent functions. Broca (4), however, in 1861,

[1] Read before the Psychological Society, at a meeting held at the Laboratory of the London County Asylums, Claybury, on April 25, 1903.

pointed out the association which exists between the power of articulate speech and the posterior part of the left third frontal convolution, and Hughlings Jackson (13) in 1864 and Charlton Bastian (1) in 1869 both wrote in favour of functional localisation.

In the year 1870 Fritsch and Hitzig (9) proved that in the dog electrical excitation of certain parts of the cortex cerebri resulted in definite and constant movements.

Ferrier (1873-6), repeated and amplified their observations on the dog, and obtained similar results in the monkey (6). Ferrier's conclusions have not entirely held good, but they have in their main essentials been confirmed and extended by numerous investigators, but especially by Horsley and Beevor (12). From the point of view of special movements, and the relation of these to special regions, numerous observations have also been made, and this subject, together with that concerning ablation experiments, possesses in itself an extensive literature. The general area, as eventually mapped out in the monkey, occupied approximately the central convolutions and the posterior parts of the frontal convolutions, together with part of the hemisphere around the upper end of median surface of the fissure of Rolando.

Horsley and Beevor also determined the area in one anthropoid ape (an orang-outang), and found that the excitable area was not continuous but was blotchy in character, and that it did not include the first frontal convolution and the upper end of the ascending parietal convolution.

Finally, quite recently Sherrington (20) has shown that in the chimpanzee the area is still further curtailed and lies entirely in front of the furrow of Rolando, *i.e.*, entirely within the frontal lobe. As has been shown by the histological researches of Campbell (5), the human excitable area probably occupies a similarly restricted region.

As long ago as 1870 Hughlings Jackson (13) drew attention to the march of the convulsion in the variety of symptomatic epilepsy which bears his name and is associated with lesions of this region; and numerous isolated clinical observations and occasional examples of cortical stimu-

lation in man have been from time to time published, and support on the whole the localisation which has been worked out by Sherrington in the chimpanzee.

The functions of this region of the cerebral cortex are not yet finally determined.

It is generally accepted that this area subserves the performance of skilled voluntary movements, and the (conscious) learning of such movements. It is, however, not yet proved whether, as is held by Ferrier (6), Schäfer (19) and others, this region is purely "motor," or at most "psycho-motor" in function, or whether with Bastian (1), Mott (16), Munk (17), and many other neurologists of note it is "sensori-motor," and is concerned with the reception of afferent impressions of general sensory and muscular origin. Experimental results and also clinical records supply such contradictory data that the question is as yet unsettled.

It is hoped that the following remarks on this question, though they somewhat anticipate a later portion of the paper, will not, in view of the exhaustive discussion to which the subject has given rise, be considered out of place. It is by no means impossible that neither the "motor," nor the "sensori-motor" view is strictly correct. It may be that the area as now defined bears a similar relationship to some region behind the furrow of Rolando to that which, for example, the visuo-psychic region of the cortex bears to the visuo-sensory. That this is not a proposition without foundation is evident from the fact that whilst Flechsig's projection system for visual impressions occupies the position of, though a greater extent of cortex than, the adult visuo-sensory area, his projection system to the central convolutions bears no such close relationship to the psycho-motor area as now mapped out (figs. 1 and 2, p. 222). In the adult this projection system more probably lies behind the furrow of Rolando than in front of it, judging from the increase of frontal development which takes place as the cerebrum attains its adult characteristics, and from the fact that half the system already lies behind the fissure. If this proposition were true, a lesion of the psycho-motor area would be homologous with one causing

word-blindness or word-deafness, &c.; in other words, this area would be the lower associational centre for kinæsthetic impressions. Such a function for this region, which possesses a direct efferent connection with the motor groups of lower neurones, seems probable in view of the evidence to be brought forward later concerning the functions of the parts of the frontal lobes lying anterior to this area. It is, to say the least, unlikely on developmental grounds that an area of the lowest order, namely, a centre of projection, would lie directly adjacent to such an important centre of association as that in the prefrontal region, and that it would possess at the same time its undoubtedly important motor functions. It is equally unlikely that such a centre, judging from our knowledge of the projection and associational areas attached to the senses of sight and hearing, would singly possess both projection and associational, as well as motor, functions.

Our present knowledge points rather in the contrary direction. As cerebral development proceeds the areas of association increase out of proportion to those of projection, and the psychomotor area becomes elaborated *pari passu* with increasing development of the animal series, and thus resembles the areas of association rather than those of projection. It is consequently not improbable that the psychomotor region is the area of association for kinæsthetic impressions, and is the forerunner of the great anterior centre of association. If this were the case its participation in such important processes of higher association as voluntary attention would be readily intelligible, and the psychomotor area would also fall into series with the posterior centres of association, and not occupy its present anomalous and uncertain position in our scheme of the functions of the cerebrum.

The present paper being chiefly concerned with the functions of the parts of the frontal lobes which lie beyond the limits of the psychomotor area, the latter will not be further referred to, and the former subject will now be introduced.

There is even less consensus of opinion concerning the

functions of the prefrontal region than exists in the case of the psychomotor area.

Ferrier (6) concluded from his experiments that the prefrontal region was the motor centre for movements of the head and eyes to the opposite side, and was hence the centre for attention.

Munk (17) divides the cortex of the monkey into some six "sensory spheres," and looks upon the prefrontal region as the "sensory sphere" of the trunk muscles. He considers that the movements evoked by stimulation of this region are due entirely to a calling up of the sensations which usually accompany these movements.

Numerous writers, notably Wundt (25) and Hitzig (11), have, however, assigned a much more important function to this region of the cerebrum, namely, that it is the centre for the highest psychic processes.

In 1895 Bianchi (2) published his researches on ablation of the frontal lobes in monkeys. His paper contains most careful notes of the psychic and physical condition of the animals operated on. He concludes that "the frontal lobes are the seat of coördination and fusion of the incoming and outgoing products of the several sensory and motor areas of the cortex." . . . "The frontal lobes would thus sum up into series the products of the sensori-motor regions, as well as the emotive states which accompany all the perceptions, the fusion of which constitutes what has been called the *psychic tone* of the individual." (BRAIN, 1895, p. 521.)

Bianchi's paper might seem to settle the question of the functions of the prefrontal region, but unfortunately his results are directly contradicted by the later experiments of Schäfer (19). "It is sufficient to cut off the part to be removed by a subcortical incision." . . . "Operating in this way I have in several cases completely removed in the monkey the whole of the inexcitable area of both frontal lobes without producing the slightest sign of the mental and intellectual dulness and alteration of character which has been regarded as pathognomonic of a lesion of this region." ("Text-book," p. 772.)

From the clinical aspect the numerous published papers

arrive at equally contradictory conclusions. For example, in 1888 Welt (23) collected fifty-nine cases of lesion of the frontal region in man. In twelve cases changes in intellect or character occurred, and in forty-seven no such changes were noted. On the other hand, in 1896 Williamson (24) analysed fifty cases of tumour or abscess of the frontal region, and found mental symptoms in forty-five cases, these being especially marked in thirty-two, and no statement of mental change in five cases. In the thirty-two there were mental dulness, loss of power of attention, loss of memory, loss of spontaneity, and the patient took no notice of his surroundings.

It is doubtful whether in this connection profit results from the collation of data from the numerous published cases of gross lesion of the cerebrum, for whilst on the one hand mental change, especially dulness and loss of memory, may be associated with, or be due to, increase of intracranial tension, on the other the absence of reference to such change may well be due, in at least many cases, to non-recognition of symptoms which would be of more significance to an alienist than to a general physician or even a neurologist.

The question of the functions of the prefrontal region has thus received no solution from the experimental and the clinical points of view, and the contradictory nature of the results is so absolute that a complete deadlock exists.

Fortunately, however, when in 1894 Flechsig (7) began his series of papers on the development of the human cerebrum by a study of the process of myelination, a new method of attacking the problem presented itself; and our knowledge of the functions of the cerebrum as a whole will have been much illumined by the extensive researches of this worker, even though his broad generalisations may not be confirmed in their entirety.

By studying the exact period of myelination of different parts of the cerebrum in the human fœtus and infant, Flechsig was enabled to divide the cortex into the two great classes of "*sensory centres*" and "*centres of association.*" The former myelinate earlier than the latter, and possess a

well-marked projection system of fibres. The latter myelinate later and are rich in long systems of fibres of association. The difference is really one of degree only, as Flechsig does not deny that projection fibres exist in the association centres, and that association systems exist in the sensory spheres.

In the whole cerebrum he described in 1898 no less than forty separate myelogenetic fields which develop at different periods.

Of the *sensory spheres* he describes four, namely, for (1) bodily sensibility; (2) visual; (3) auditory; and (4) olfactory and gestatory sensations. (1) has eight separate myelogenetic fields, and each of the others has three each.

Of the *centres of association* he originally described four, namely, the frontal, the parietal, the temporal and the insular. Later he combined the temporal and parietal centres into one, the great posterior centre of association. In 1900, however, owing to his discovery of a centre of projection in the gyrus subangularis, he again separated these centres.

In the temporal and parietal centres of association there exist, according to Flechsig, peripheral zones, which develop earlier, and central zones, which develop later; the former adjoin the sensory centres, and are united to them by numerous arcuate fibres.

In the frontal centre of association similar zones exist, but their disposition is much more complex.

The insular centre of association, and also that in the precuneus, consist of peripheral zones only. Flechsig is of opinion that the peripheral zones may be intermediate types between the central territories of association and the sensory projection spheres.

Hence of the centres of association the *frontal* exhibits the greatest complexity, the *temporal and parietal* are intermediate in structure, and the *insular* and that in the *precuneus* are the least complex of the types (see figs. 1 and 2).

Flechsig's views on the functions of the central territories of the areas of association are as follows:—"The

central territories of the zones of association (especially the middle of the angular gyrus, the third temporal convolution, and the anterior half of the second frontal

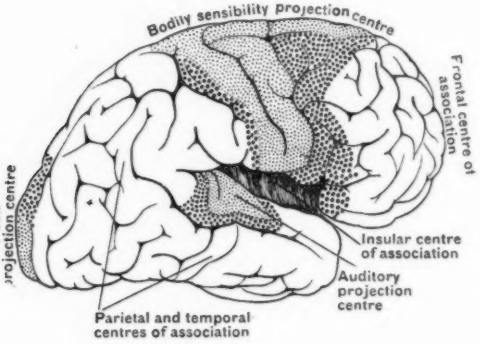
Bodily sensibility projection centre
Frontal centre of association
Visual projection centre
Insular centre of association
Auditory projection centre
Parietal and temporal centres of association

FIG. 1 (AFTER FLECHSIG).

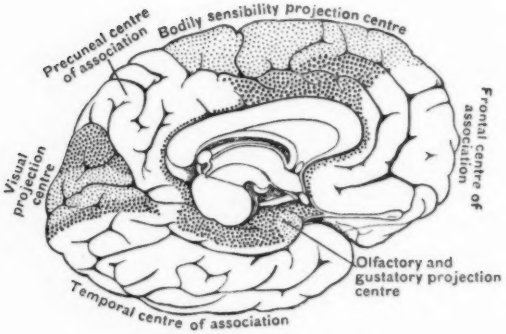
Precuneal centre of association
Bodily sensibility projection centre
Frontal centre of association
Visual projection centre
Olfactory and gustatory projection centre
Temporal centre of association

FIG. 2 (AFTER FLECHSIG).

In these figures the "centres of projection" and "centres of association" are mapped out according to Flechsig. The small dots are placed in the chief focus of each centre of projection. Around these chief foci are the regions to which a smaller number of fibres of projection pass; these are indicated by larger dots. These figures, which represent the different cortical spheres as they exist in the human fœtus and infant, should be compared with the areas which have been fully defined in the adult cerebrum, *e.g.*, in the case of the psychomotor area, papers by Campbell (5) and Sherrington (20), and, in the case of the visual projection area, by the author (3). See also p. 217 of the present paper.

convolution) are apparently the nodal points of the long systems of association, whilst the peripheral zones only feebly show these characteristics."

"The central territories are terminal territories: they are essentially characteristic of the human brain. Isolated destruction of these is never accompanied by phenomena pointing to disturbance of motion or sensation. Motor phenomena, which occasionally accompany lesions of these territories, must be interpreted as *actions à distance*."

"The central territories of the zones of association are in more or less direct relationship, each with several sensorial zones, some with all."

"After their bilateral removal intelligence is affected, and especially is association of ideas interrupted. These central territories are thus probably of great importance for the exercise of intellectual activity, for the formation of mental images composed of several sensory qualities, for the accomplishment of acts, such as the naming of objects, speaking, &c. These functions are especially interfered with in affections of the posterior centres of association. Clinical observation establishes this fact, and thus justifies the legitimacy of our division of the cerebral cortex into sensory centres (centres of projection) and centres of association." (*Archiv de Neurologie*, ii., 1900, pp. 337-8.)

Flechsig's views have been severely criticised by Hitzig (11), who, however, is disposed to admit their general truth in a less positive form; and by Vogt (21), who directly contradicts many of Flechsig's conclusions, and especially the thesis of the constancy and regularity of myelination of the different systems of fibres. He is inclined to look upon the majority of myelinated fibres as projection fibres. Monakow (15), in his criticism, draws attention to the small proportion which the projection fibres form of the total mass of cortical fibres in any of the convolutions, and he thinks that it is not possible, however roughly it be done, to define the regions which are poor in these fibres and those which are abundantly supplied with them. Sachs (18), whilst admitting that the sense centres are sharply marked off at an early period

from the rest of the cortex, thinks it impossible to prove later, when myelination has advanced, that medullated projection fibres do not pass to the centres of association.

On the whole, however, Flechsig's conclusions in their essential features have been widely accepted, and whilst the projection areas, as has been already pointed out, probably occupy neither the identical position nor the same extent of cortex in the adult brain that they do in the fœtus and infant, it may probably be assumed that a great parieto-temporal association centre exists posteriorly, and a more complex prefrontal association centre anteriorly, the insular and precuneal centres being less complex in type, and probably of less importance, than either of the former.

With reference to the functions of the two greatest centres of association, a large body of neurologists, notably Bastian, Hughlings Jackson, Schäfer, and Flechsig himself, are of opinion that gross mental disabilities are more likely to occur in lesions of the posterior centre than of the anterior, whilst Wundt, Hitzig, Ferrier, &c., hold the opposite view.

From the neurological aspect, especially when considering the different varieties of sensory and motor aphasia, the former view is doubtless true, but premising that the posterior centre were concerned with lower associational processes only, general mental disability would still be evident in cases of gross lesion of the hinder part of the hemispheres, as the patient would under these circumstances be unable in many cases to produce satisfactory evidence of general mental soundness. In many other cases, also, such an entire disturbance of perceptive and ideational processes as occurs, would be not unlikely to cause too great a strain on the higher associative functions, and to directly result in the development of symptoms of true mental alienation. This is rendered probable by the fact that no less than one in every 293 of the general population[1] is at present suffering from mental alienation, and that the proportion of potential psychopaths is very much greater.

[1] Report of the Commissioners in Lunacy for 1902.

Hitherto in this connection very little attention has been paid to the cerebral lesions associated with mental disease, though it is very generally recognised, by alienists, that in chronic general paralysis and chronic senile dementia severe prefrontal wasting exists. In the case of the former variety of mental alienation, however, the question is obscured by the thesis that previous syphilitic infection is the chief, if not the essential cause of the disease; and in the case of other varieties of mental disease, whilst their morbid histology has for many years attracted much attention, relatively little care has been bestowed on the *correlation* of the morbid anatomy and clinical features of the different types of mental alienation.

During the past four years the writer has made a careful study of this question, and he has arrived at the conclusion that the great anterior centre of association is the region of the cerebrum which is primarily affected in mental disease, all the neighbouring or bordering regions being concerned to a less extent, probably from chronic atrophy of the related systems of fibres of association.

The summarised evidence on this point which will now be brought forward will be considered from the points of view of (1) *morbid anatomy*, and (2) *general histology*. Fuller details on many points not directly bearing on the subject under discussion are contained in the paper referred to below (3).

(1) Morbid Anatomy of Mental Disease.

In 200 cases of mental disease, recently reported by the writer in the second volume of the *Archives of the Laboratory of the London County Asylums*, it has been demonstrated that the amount of cerebral wasting and the associated morbid changes inside the cranium in these cases vary directly with the amount of dementia existing in the patients. The relationship is much more absolute than might at first appear, probably owing, in the majority of cases of insanity, to a more or less complete removal of the products of degeneration by the time of death. This relationship is less

constant in dementia paralytica (general paralysis of the insane), for whilst in chronic cases the wasting may be more profound even than in corresponding cases of severe dementia, associated with senility and gross vascular degeneration, in acute and rapid cases of the disease, owing to incomplete removal of the products of degeneration, much less wasting exists. The writer has, however, seen several cases in which macroscopic and microscopic *acute* changes were as marked as is the wasting in advanced chronic cases (see later).

In many cases associated with severe vascular degeneration a further complicating factor exists, namely, the presence of local or general atrophies which are directly of vascular origin.

On the whole, however, as has been shown in a previous paper, the relationship is remarkably exact, and the regions of relative wasting can be determined with considerable accuracy. Taken generally, for individual variations exist, the *regions of wasting*, from observation of several hundred cases, are as follows :—

(1) The greatest amount occurs in the prefrontal region (anterior two-thirds or so of the first and second frontal convolutions, including the neighbouring mesial surface, and the anterior third or so of the third frontal convolution).

(2) The wasting is next most marked in the remainder of the first and second frontal convolutions. [In dementia paralytica Broca's convolution should, as a rule, be included here and (2) and (3) should follow (4).]

(3) It is, perhaps, next most marked in the ascending frontal and Broca's convolutions, though this grade should, in many cases at least, follow (4).

(4) It is next most marked in the first temporal convolution and the insula, and in the superior and inferior parietal lobules. In practically all cases it is more marked in the two former than in the two latter.

(5) It is least marked in the remainder of the cerebrum (including the orbital surface of the frontal lobes), particularly the inferio-internal aspect of the temporo-sphenoidal lobe and the posterior pole of the hemisphere.

In the experience of the writer exceptions to this general order are invariably due to vascular or traumatic causes, and should, therefore, be excluded from the ordinary and normal wastings of dementia.

Apart from the necessarily excluded abnormalities of development, which are of vascular or traumatic origin, the *degrees of under-development* follow the order given above, at least as regards (1) and (2). A further statement regarding macroscopical detail cannot be made, as it is more usual to find small and simply convoluted cerebra than brains with average but small convolutions, and it is relatively rare to meet with under-developed brains of average convolutional complexity which show a decrease reasonably comparable with the marked wasting which occurs in severe dementia. The subject of under-development will be more completely considered under the heading of general histology.

It is desirable here to state that from the histological standpoint Watson (22) has recently arrived at almost exactly similar conclusions regarding the comparative degrees of affection of the different regions of the cerebrum in several cases of juvenile general paralysis.

It is unnecessary here to give instances of the morbid conditions under consideration, as numerous examples, with in some cases illustrations, of the respective degrees and types of wasting which occur in mental disease are recorded in the paper already referred to. The following case of dementia paralytica, which has recently come under the notice of the writer, is, however, deserving of record here, owing to its almost unique character, and also to its direct bearing on the subject of the present paper. As will be seen in the illustrations (plate), the association centres of Flechsig are mapped out in this case as clearly almost as in the diagrams (fig. 1 and 2, p. 222). The anterior centre is grossly wasted; the psychomotor area shows some but much less wasting; the great posterior centre of association (temporal and parietal), the insular, and the precuneal, are acutely changed, as is evidenced by the decortication which in this case is *not* a *post-mortem* phenomenon; and the visual projection area is practically or entirely intact. The

FIG. A.—Photograph of the outer surface of the left hemisphere of Case G. F. (p. 229).

The anterior centre of association is grossly wasted. The psychomotor area shows some, but much less, wasting. The temporal and parietal and the insular centres of association are acutely changed (*post mortem* four and a half hours after death). The upper part of the temporal centre of association, and the anterior part of the parietal, show less acute change and more wasting. The visional projection centre is intact.

This illustration should be compared with the diagram on Fig. 1 (p. 222), and also with the plates in the author's paper in the *Archives of Neurology*, vol. ii.

FIG. B.—Photograph of the inner surface of the same hemisphere.

The anterior centre of association is both grossly wasted and acutely changed. The temporal and præcuneal centres of association are acutely changed. The visual projection centre is intact. There is wasting of the marginal convolution and acute change of the callosal convolution. The acute change in the prefrontal and callosal regions is in the situation where the pia mater of the two hemispheres lies in contact below the falx cerebri, and where, in general paralysis, the earliest and also the most advanced changes are visible.

This illustration should be compared with the diagram on Fig. 2 (p. 222).

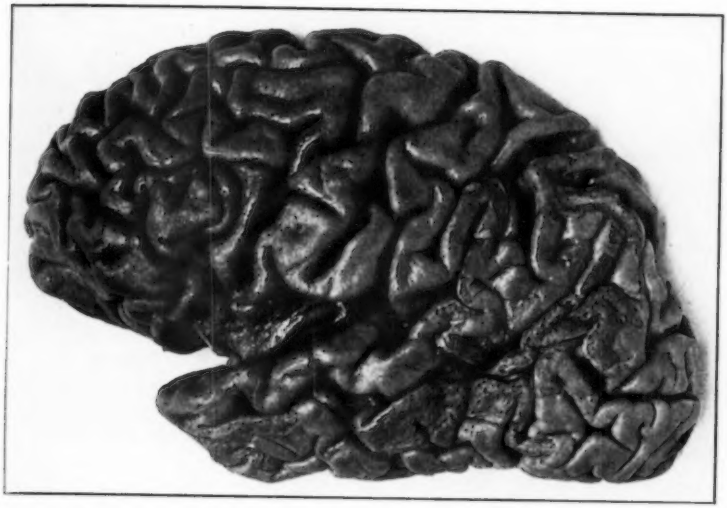

FIG. A.

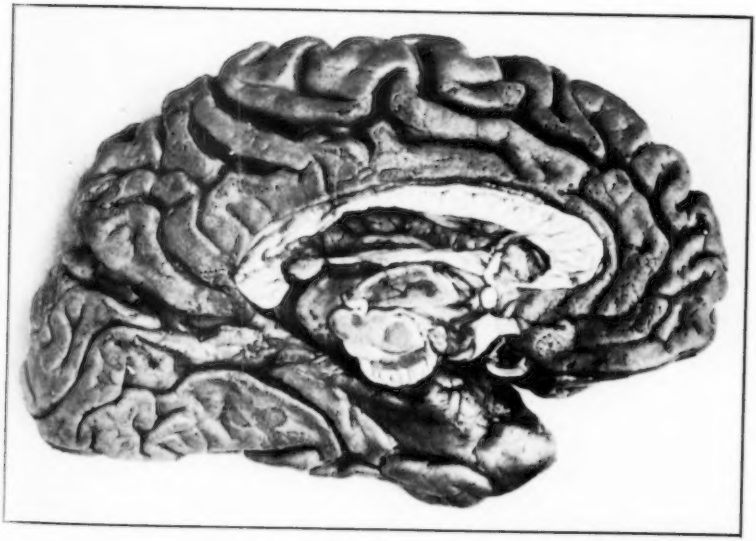

FIG. B.

writer has now seen a sufficient number of similar cases to satisfy him that this condition, though unusual, is normal to a particular type and stage of general paralysis of the insane, granted that the patient dies at the proper time from an intercurrent complication. Such cases illustrate more clearly than do those of any other variety of mental alienation the primary affection of the anterior centre of association, which is followed later on by decay of the posterior centres of association, the projection centres, (especially the visual), which are the first to develop, still remaining relatively unaffected. As a rule the psychomotor area is not markedly affected, though in certain cases commencing with numerous convulsions (*e.g.*, Case 20, in Part II. of the paper above referred to), this region may be early involved in the process of dissolution.

Whilst it is to be regretted that more extensive clinical notes of the following case were not obtained during the four weeks the patient was in the asylum, this was unavoidable owing to the large number of cases under observation, and the impossibility of taking really complete notes even of every severe case of general paralysis.

G. F., admitted March 31, 1903. Died April 28, 1903. Age stated to be 26 years. Single. Private, Rifle Brigade. No satisfactory family or personal history, though heredity of insanity was denied. Was admitted under the following certificate:—"Low demented state. Dirty in habits. Incoherent. Has convulsive seizures and paresis of limbs. Right pupil much dilated. Suicidal. Thinks people are trying to do him an injury and otherwise annoy him. Moping and inert. Sits for hours muttering incoherently. Memory quite fogged."

Physical condition on admission.—Tattoo marks on arms. Tongue tremulous. Incontinence of urine. Pulse small and irregular. Heart weak. Right pupil larger than left. Both irregular and neither reacts to light or accommodation. Both undergo irregular rhythmic movements and become eccentric at irregular intervals. Knee jerks, + +. All the limbs undergo chronic contractions and sometimes enter a pseudoclonus. Continual masticatory movements of lower jaw.

Mental condition on admission.—Patient lies in bed with his limbs flexed and rigid. He does not understand anything when

he is spoken to but at times makes inarticulate noises, especially if he is touched. He willingly takes his food from a feeder. He seemed to recognise his brother when he visited him, and tried to speak but could not be understood.

April 15, 1903.—His pupils vary as on admission and his limbs vary in position and degree of rigidity, but apparently quite irregularly. No data can be obtained from him regarding his mental condition, but he tends to resist examination though he is otherwise quite helpless.

April 28, 1903.—Died of right lobar pneumonia.

Post Mortem (Four and a Half Hours after Death).

Dura mater natural. Subdural deposit:—A non-hæmorrhagic film, the thickness of tissue paper on the right vault. Remarkable excess of subdural fluid. Pia:—Extremely opaque, and almost universally thickened. Marked mid-line pre-frontal adhesions below the falx cerebri. Great excess of sub-arachnoid fluid, especially in the pre-frontal region. Lateral ventricles immensely dilated, and granular Third ventricle granular. Fourth ventricle very granular throughout, but especially so in calamus. Vessels apparently natural. Weight of encephalon 975 grammes. Right hemisphere, unstripped, 395 grammes. Left hemisphere, unstripped, 395 grammes, ditto, stripped, 350 grammes. Cerebellum, pons and medulla 157 grammes. There is extreme wasting of the prefrontal region, relatively little of the psychomotor area and the post-central convolution, and none of the occipital pole and the visuo-sensory area. The pia-arachnoid strips very readily over the frontal region. There is extreme decortication of the parietal, temporal, and insular association centres externally and of the precuneus and the inner part of the temporo-occipital region internally. There is also decortication scattered irregularly throughout the callosal convolution, but the visuo-sensory area is practically free from decortication. There are no absolute *post-mortem* signs of syphilis, but the density of the liver, spleen, and kidneys is increased.

Cause of Death:—Right lobar pneumonia. General paralysis.

(2) General Histology (Normal and Morbid) of the Cortex Cerebri.

Regarding the special cellular elements of the cortex cerebri which are affected in mental disease, it is a well-

known histological fact that the most commonly affected cells are those of the pyramidal layer. The writer is, however, able, as the result during the past seven years of an extensive study of the cortex cerebri by the method of micrometric measurement, to make a number of very definite statements concerning the development and retrogression of the cellular elements of the cortex cerebri, both in normal individuals and in the subjects of mental disease. His conclusions are derived from the examination of many thousands of micrometric measurements of the different cell layers of the cortex, which have been published in more or less complete detail in previous papers.

The cortex cerebri in the regions referred to above as the centres of association of Flechsig, consists in the adult of five primary layers; of these, two, the first or superficial, and the fourth, are essentially nerve fibril layers. The remaining three, namely, the second, the third and the fifth, containing respectively the pyramidal, the granule, and the polymorphic cells, are essentially nerve-cell layers. The writer makes no attempt to sub-divide the second or pyramidal layer, which is structurally single, into layers of small and large pyramids, and he considers that the ganglionic or Betz cells, which lie in certain regions of the cortex cerebri, belong to the fourth layer or inner line of Baillarger, that they are homologous with the solitary cells lying in this layer in other regions of the cortex, and that they are not "pyramidal cells" at all.

In the investigation from which the immediately following data are derived, the cortex made use of was taken from, as it were, the focus of the wasting referred to above, namely, from the anterior pole of the hemisphere in the region of the second frontal convolution, and at right angles to the (constant) transverse fissure of Wernicke. The region employed was as far as possible of the same relative size in all the brains examined; serial sections were made, and as far as possible mathematical accuracy was attempted, both in the choice of sections and in the micrometric measurements prepared from them.

In the prefrontal region of the cerebrum lamination

begins about the sixth month of fœtal life by the separation off of the fifth or polymorphic layer of nerve-cells, and the fourth layer (of fibres) or inner line of Baillarger, and both these layers are very little below (about three-quarters of) the normal depth almost from the first appearance of the lamination. The third layer, or layer of granule cells, next develops, and at the periodr eferred to is only about one-half the normal depth. At this period the second layer, or layer of pyramidal cells, is only one-fourth of the normal depth.

The development of the layers of the cerebral cortex thus takes place first in the deepest part of the originally indifferent neuroblasts and the more superficial layers develop later; in other words, the process takes place from within outwards.

At birth the second or pyramidal layer is but little more than one-half of the depth of the adult layer, the third or granule layer has now become three-fourths of the adult depth, and the fourth and fifth layers are rather more than three-fourths of the adult depth.

In the normal adult prefrontal cortex the depth of the third, fourth, and fifth layers is practically constant in different individuals, but differences exist in the depth of the second or pyramidal layer of nerve-cells. This layer of cells is, of all the cortical layers, the easiest to measure accurately, and hence these differences actually exist.

In congenital amentia (idiots and imbeciles) degrees of under-development of the prefrontal cortex exist which vary inversely with the mental power of the individual concerned.

In chronic and recurrent insanity without dementia, the third, fourth, and fifth layers of the prefrontal cortex are approximately of the normal adult depth, but degrees of under-development of the pyramidal layer of nerve-cells exist.

In dementia, and in dementia paralytica, degrees of wasting or retrogression exist in the layers of the prefrontal cortex which vary directly with the amount of dementia present. When the mental power of the patient is that of the new-born child all the cortical layers are, as regards depth, approximately in the same condition as in the

latter. This fact is graphically shown in the following table, in which the results of micrometric examination of the prefrontal cortex of certain cases is summarised :—

Type of case.	Layer I. Superficial nerve fibrils.	Layer II. Pyramidal cells.	Layer III. Granule cells.	Layer IV. Inner line of Baillarger.	Layer V. Polymorphic cells.	Total depth of cortex in millimetres.
	mm.	mm.	mm.	mm.	mm.	mm.
Normal (average of cases 1, 2 and 3)	·3005	·8312	·2288	·2303	·3098	1·9006
Still-born infants (average of cases 6 and 7)	·2024	·5047	·1661	·1663	·2270	1·2665
Gross dementia (average of cases 19 and 20)	·2858	·6568	·1748	·1581	·2130	1·4885

When the neuroglial and vascular proliferation which exists in the first two layers of the third series is allowed for, the last two series show a marked resemblance to one another, as do the corresponding mental conditions; and as a child under the age of a few months is still relatively helpless and mindless, an even closer parallel of measurements might be made than the above. The two series given, however, representing as they do the condition of the cortex at *birth*, when mentation is about to begin, and *at death in the final stage of primary decay of the prefrontal cortex*, with the mind practically gone, are sufficiently striking.

The above data concern the prefrontal cortex in normal individuals of different ages and in the subjects of the different types and grades of mental disease.

The writer has, however, also mapped out and similarly examined the whole visuo-sensory region, or region of the cortex to which visual impressions primarily pass, and also a considerable portion of the adjoining cortex. The following data thus serve to illustrate similar features of the projection and posterior associational areas of the cerebrum.

In the visuo-sensory cortex (projection centre) the pyramidal layer of nerve-cells is developed earlier than is the corresponding layer in the surrounding visuo-psychic cortex (centre of association). For example, in children of one and three months respectively, the pyramidal layer of nerve-cells

in the visuo-sensory region is relatively little below the normal adult depth, whilst in the surrounding cortex in the child of three months the pyramidal layer is much below the normal adult depth, and in the child of one month it is less than two-thirds of the latter.

This fact constitutes an important proof of the associational function of the pyramidal layer of the cortex surrounding the visuo-sensory region, and it agrees with Flechsig's discovery that the fibres passing to the primary visual area myelinate earlier than those to the surrounding cortex. The fact also that the pyramidal layer of nerve-cells of the visuo-sensory region is in the adult only five-ninths of the depth of the corresponding layer in the surrounding cortex, also supports the thesis of the associational function of this layer in the latter region of the cerebrum.

On the other hand, in the dements examined, though the amount of dementia differed in the different cases, the decrease present in the pyramidal layer in all the cases was practically the same and very slight, in this differing from the results obtained in the prefrontal region, where it varied with the amount of dementia. The pyramidal layer was also actually as much, and relatively to its depth more, decreased in the visuo-sensory region than in the visuo-psychic. This agrees strikingly with the fact that in dementia the loss of recognition and of recent memory is more obvious than that of remote memory, though naturally little stress can be laid on this detail. It is certain, however, that whilst in *all* cases the depth of the pyramidal layer of nerve-cells in the prefrontal region varies directly with the mental powers of the individual, this is not the case in the primary visual and neighbouring visuo-psychic regions, though here, as has already been stated, the pyramidal layer develops earlier in the former than in the latter region.

From the above data the following conclusions may be drawn concerning the cortical cell layers:—

(1) *The second or pyramidal layer of the cortex cerebri.*

(*a*) *The prefrontal region.*—The pyramidal layer is the last cell-layer of the cortex to develop during the process

of lamination, and it is also the first to undergo retrogression in dementia. It is the only layer which appreciably varies in depth in normal brains; the degree of its development in normal fœtuses and infants, and in congenital aments of every grade, varies directly with the mental power of the individual; and the degree of its retrogression in demented patients varies directly with the amount of dementia existing in the respective patients. (*b*) *The visuo-psychic region.*—The pyramidal layer reaches practically the same adult depth as in the prefrontal region, but it does not vary in depth according to the degree of dementia, though a small and practically constant decrease in depth is evident. This layer develops much later than does the pyramidal layer in the visuo-sensory region, and in a child of one month it is less than two-thirds of the adult depth. (*c*) *The visuo-sensory region.*—The pyramidal layer in this region is in the adult only about five-ninths of its depth in the regions above referred to. It, however, develops much earlier, being in infants of one and three months very little below the adult depth.

The pyramidal layer, therefore, subserves the "psychic" or associational functions of the cerebrum. This is preeminently the case in the prefrontal region, less so in the visuo-psychic region, and least of all in the visuo-sensory region. These three regions are therefore of different grades in the hierarchy of cerebral function.

(2) *The third, or granule layer,* is developed before the pyramidal layer. In the primary visual area the optic radiations end in the midst of the hypertrophied and duplicated granule layer. *This layer, therefore, probably,* reasoning by analogy, *subserves the reception or immediate transformation of afferent impressions, whether from the sense organs, or from other parts of the cerebrum.*

(3) *The fifth or polymorphic layer* is the first layer to be differentiated during the process of lamination, and it is the last to fail in the retrogression of dementia. A decrease in this layer exists in extreme aments (normal or otherwise), and in dements who are unable to carry on the ordinary animal functions, such as attending to their own wants, &c.

This layer, therefore, probably subserves these lower voluntary functions of the animal economy.

The *first and fourth layers* of the cortex cerebri, being primarily cell-process layers, occupy a different position. It is, however, of great interest that these layers are laid down in neuroglia long before fibrils can be detected in them, *e.g.*, in the prefrontal region the first layer exists as an irregular line of varying thickness in the fourth month of fœtal life, and the fourth layer develops during the sixth month of fœtal life. It is not intended by the above to deny that the relatively small number of cells, which, in the adult state of the cortex especially, are contained in these layers, may and probably do possess important though minor functions in the processes of cerebration. In the psycho-motor area, for example, the Betz cells, which really belong in the opinion of the writer to the fourth layer or "inner line of Baillarger," and are therefore not "pyramidal" cells at all, constitute the origin of the important efferent tract for skilled voluntary movement. Probably the "solitary cells" of Meynert in the occipital cortex possess a somewhat analogous function, and perhaps the same may be stated concerning the more or less pyramidal-shaped cells which lie in the fourth layer or inner line of Baillarger, in other regions of the cerebrum. It is probably hardly justifiable to endeavour to assign a function to the few cells which lie in the first or superficial layer of the cortex cerebri, but perhaps, reasoning on general grounds, it is not unfair to suggest that they possess associational functions similar to those of the pyramidal layer, above which they lie, and with which, in order of time, they appear to be developed.

Concluding Remarks.

The evidence above adduced, which is derived from a study of the general histology of the cerebral cortex in normal individuals, and of the morbid anatomy of the cerebrum and the general histology of the cortex cerebri in the subjects of mental disease, points, in the opinion of the writer, irresistibly to the conclusions that *the anterior centre*

of association of Flechsig is the region concerned with attention and the general orderly coördination of psychic processes, and that *the cellular elements throughout the cortex which are especially concerned in the performance of associational functions are those of the pyramidal layer of nerve-cells.*

From the standpoint of morbid anatomy the writer considers it proved, with the necessary restrictions already referred to, that the amount of cerebral wasting present in any case varies directly with the degree of dementia existing, and that this wasting has its maximum focus in the pre-frontal region.

Still more definite and positive results have, however, followed his study of the general histology of the cortex cerebri by the micrometric method. The pyramidal layer of nerve-cells develops *pari passu* with the development of the pyschic powers of the individual, whereas the other cell-layers of the cortex develop earlier and soon reach their adult depth. In the visuo-sensory area (a type of the centres of projection), the pyramidal layer develops earlier than in the visuo-psychic region (a type of the posterior centres of association) and even soon after birth is relatively little below the adult depth, which is only about five-ninths of the depth of the layer in the centres of association. In the visuo-psychic region the pyramidal layer attains the same depth as in the pre-frontal region, but the decrease in depth in dementia is small and does not vary with the degree of dementia. Further, in the pre-frontal region, in the different types of mental alienation grading from the idiot to the chronic or recurrent lunatic without dementia, the pyramidal layer exhibits degrees of underdevelopment which vary inversely with the mental power of the individual: and it is the only cell layer of the pre-frontal cortex which varies appreciably in depth in normal individuals, although of all the layers of the cortex cerebri it is the easiest to measure accurately, which fact proves that these differences are not accidental. Finally, in dementia (which term is used to connote the mental condition of patients who suffer from permanent psychic disability) the amount of thinning of the pyramidal layer of nerve-cells in the pre-frontal region varies directly with the degree of dementia present.

The writer hence considers it proved that *the great anterior centre of association lying in the prefrontal region is underdeveloped on the one hand in all grades of primary mental deficiency, and on the other undergoes primary atrophy* pari passu *with the development of dementia; it is therefore the region of the cerebrum which is concerned with the performance of the highest coördinating and associational processes of mind.*

From the point of view of pure alienism this thesis receives illustration in the impulsive and irrational acts of many types of lunatic, and in the mechanical and stereotyped mental processes of the dement. Further illustrations are found in the absence of self-consciousness (as regards the presence of others), in the extreme vanity, and in the callous and selfish behaviour, which are common characteristics of the insane; but especially by a study of the development of delusional states. The delusional state is a secondary development, having as its basis a deficient power of coördination of psychic processes, and an inability to correctly interpret and suitably coördinate present experience in the light of past. In the paranoiac the capacity to develop it is present long before accident of environment determines the particular variety, and also the content of the, at first temporary but finally permanent, delusional state which ensues. The primary causal factor, however, progresses more rapidly than does the delusional state, and hence in some cases mild and in others more marked dementia eventually becomes evident. In a typical example this may ensue in the absence of hallucinations, in which case it may be presumed that there is only a relatively slight affection of the lower centres of association. This variety of mental disease may thus, perhaps, be considered to be in some respects a connecting link between, on the one hand, the different grades of primary mental deficiency, and on the other, the cases of more or less simple progressive dementia.

It is not desirable to enter further into this matter in the present paper, but it has been thought worth while to introduce the above remarks in illustration of the writer's

thesis concerning the functions of the pre-frontal region of the cerebrum.

That the study of gross lesions of the cerebrum has resulted in such contradictory conclusions concerning the functions of this region is readily explicable on the ground that such lesions, whether the symptoms resulting from them be direct or indirect or due to a general disturbance of the cerebral functions, do not necessarily interfere seriously with the functions of the complex associated groups of neurones focussed in the pre-frontal region. It is probably only when the whole complex system of neurones is under-developed or when it is undergoing general primary decay, namely, in the different types and degrees of mental alienation, that a satisfactory insight into its functions can be obtained; and even under these conditions, owing to the highly intricate systems of association which connect the pre-frontal region with the other parts of the cerebrum, the resulting lesion is not pure, but is merely focussed in the anterior parts of the frontal lobes.

Again, that the results obtained from experimental interference with the pre-frontal region in monkeys are equally contradictory is explicable on similar grounds. If the lesion were complete, owing to the close association between, if not the actual overlapping of, the pre-frontal and psychomotor regions, motor phenomena would necessarily invalidate the results, and any less extensive lesion cannot, in the opinion of the writer, be considered conclusive. Still more important, however, is the immense difference which exists between the psychic powers of even the highest monkeys and those of human beings; and apart even from this difference, it is probable that a general primary decay of the pre-frontal associated systems of neurones is the only condition which, even in the monkey, could enable a satisfactory solution of the problem to be obtained.

Our knowledge of the structure and mode of development of the cerebral cortex in the different members of the animal kingdom is still in its infancy and it is probable that future research in this direction will be fruitful with reference to the functions of the cerebrum both in man and in the lower animals.

The writer desires in conclusion to express his indebtedness to Dr. F. W. Mott, F.R.S., Pathologist to the London County Asylums, for so kindly giving him permission to make use of the material collected during the three and a half years he acted as his assistant; and also to his other late chief, Dr. Robert Jones, Superintendent of the London County Asylum, Claybury, for affording him such ample opportunities of increasing his experience during the same period.

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CLINICAL STUDIES.

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I.—On "Dreamy Mental States" as a Permanent Condition in Epileptics.

I recently published a statement on dreamy mental states[1] enlarged by my own observations. This condition, which has hitherto hardly been recognised on the Continent, has been thoroughly studied in England, particularly by Hughlings Jackson and Crichton Browne. Through these studies my attention has been directed to a form characterised by its remarkable course, which, as far as I can see, has not been mentioned by these authors.[2]

I should like to offer this paper as a tribute of respect to the Master of Neurology, Hughlings Jackson, in this Journal, which contains his works on the subject.

Before entering upon the clinical description it will be well first of all to state the psychopathical symptoms under which, according to the observations of the above-named authors and my own, the dreamy mental states run their course. One of the most important phenomena is the circumstance that the patients—even highly-cultured ones—state that the conditions are so peculiar, so obscure and of so rapid a course that they cannot describe them at all;

[1] *Prag. med. Wochenschrift*, 1901, Nos. 39 and 40.

[2] Having said in the text that I had found nothing in literature similar to what I am about to describe, that observation is to be corrected in so far as Crichton Browne in his Cavendish Lecture "On Dreamy Mental States," page 17, mentions that the entire mental disturbance of one of his patients consisted in a connected series of dreamy states, thus seemingly in a condition that might be similar to that which I am going to describe, but the further description of this condition demonstrates that it was rather a question of claustrophobia and not of the typical phenomena of the dreamy states, as in my cases.

they complain of a sensation of being somewhere else, or of a double consciousness, of a loss of consciousness of personality; they describe the condition as a return to youth, as the recurrence of an old dream, as a loss of the perception of exterior objects, of their own corporeality, or of a disturbance of the latter; they complain of sudden sensations of despair, or a feeling that the judgment day has come; I, myself, in addition heard complaints of impetuous flight of thought, though, nevertheless, it is possible for the mental faculties to be maintained meanwhile, so that the condition of the environment seems unchanged. Further, there are terrible, horrible thoughts that cannot be put into words. It is also to be borne in mind that Hughlings Jackson called attention to these states being connected with an aura of the sense of taste or smell; this I can confirm, as such patients occasionally also complained that during and often for long after the attack they could neither smell nor taste anything, that everything appeared to them tasteless and odourless.

Before communicating two cases which demonstrate that the conditions described above may also occur chronically, unmistakeably as permanent conditions, and that thereby not only the intervening periods, but epilepsy itself, assumes an entirely different stamp, I may remark that the statements made by the patients in their diaries, which were placed at my disposal, originate from themselves. I prefer in such cases this method of description to personal examinations, as in their diaries the patients give freer and more unvarnished impressions than at a medical examination; from these diaries there might also be elicited other peculiarities in this category of patients.

Case 1 is that of a young lady, 30 years of age, who has frequently been in my clinic in consequence of post-epileptic conditions of excitement. There are several cases of epilepsy in her family; she herself has been epileptic since her twelfth year; in addition to the symptoms, associated with the typical attacks of convulsions, she occasionally also suffered from uncontrollable impulses, as, for instance, the desire to plunge a knife into the body of one of her relations. During recent years this patient

had had fewer attacks in consequence of suitable treatment, but instead of, and in addition to attacks of petit mal, there set in peculiar conditions of a psychopathical nature, which will be described, and which were so torturing that the patient longed for the former condition with the severe convulsive attacks. She, moreover, complained of frequently recurring painful disturbances of sensibility in the left half of the body which were introduced by an olfactory manifestation. She accurately stated that the conditions in the head had commenced since the attacks of convulsions had become less frequent; the observation of these had incidentally exhibited a prevalence of the twitchings of the muscles of the body on the left side.

The following extracts from the diary of the patient during the frequently lengthy intervals between the attacks, show her peculiar mental condition.

January 22. The billows beat together over my head. Again and again innumerable nervous conditions instead of attacks, these strange guests within me. I am becoming sucked dry as if by the leeches within me. From early dawn, when I open my eyes, until late in the evening, the whole day through this chase in me, this hunt in my inside goes on, it is awful, I am already so hunted inside, I write hardly anything of it, it is like a drop in the sea, compared to what I feel. I cannot describe it, to me it is incomprehensible what is going on internally, uninterruptedly; I force myself to other things . . . I write; but the actual, the real, how and what is with me, I do not write, cannot write what I should like to write. That cannot be described! I am in my mind and altogether hardly half of myself. And, to-day as always, I am not right inwardly, not comfortable. I am always so confused inside.

January 30. But in my head something that I cannot describe. I am incomprehensible to myself, and have then always forgotten everything. . . . And thus often in the night, I thought that I might sleep, I was sleepy, then it overcomes me, I do not know in and out, not where and what it is, this mental and inward jumble. How I felt the whole night through and early morning, it is hard to describe. . . . Since to-day's attack I am confused, muddled up in my head. I constantly feel as if an attack were coming on. In the evening I had a long strong sensation, and afterwards I kept on feeling more disturbed, confused, indistinct in the head. In the night, too, on awaking and throughout sleep I am conscious that I am not all right in my head.

March 11. Early Saturday, while still in bed, I had a long

strong sensation; I was as if only half conscious, my head was all confused, I was not quite in my right mind and there was something rigid, convulsive in the arms by the wrists, by the pulses; I did not seem to have my wits; as if, instead of the two sensations, severe heavy attacks had been coming on and had stuck fast in me. In the morning, I felt as if I ought to allow my head to have a long rest (but I do not know how) from great, great exertions (which?). Mid-day. I was not excited either inwardly or outwardly; what was happening in my head? It was as if something dreadful, unforeseen, must or should break out in my head. I said nothing about what was going on in my head for so long, indeed I could not properly. What is the matter with my head? I cannot describe at all what is going on so long, so often in my head. . . . I am mostly in a sort of stupor, that is for several weeks I appear vacant to myself. I feel it, I believe often that I can perceive what it is that is coming over me, as if, may God forbid, I am not far removed from becoming an idiot. . . . I am not right in my head. Not yesterday, not to-day, not by day, not by night, in sleep, in dreams, in waking, it cannot be described. If a sort of sadness falls on me I can say I am still a little well. . . . I cannot describe it, the terrible aversion that overcomes me, is not the proper expression. This fatigue in the head. . . . I am so entirely exhausted, not only to-day, as if something gloomy gained more and more power over me. Something overpowers my head, I know not what, that gains power over me and I am weak. . .

Wednesday. I can trace such a terrible, oppressive, disagreeable confusion in my head during sleep, throughout all jumbled dreams, not only last night and the night before, but quite often. Such a confusion, such pell-mell. Something oppressive, so melancholy. I also have it before going to sleep, on waking, this terrible not to be described, that robs me entirely of clearness.

October 24. In the evenings before I go to sleep, it is so obscure, so unpleasant, so uncomfortable in my head, I can scarcely grasp a correct thought. There is something not right in my head, I feel it through my sleep. And then when I wake early I feel as if I were coming into a quite different, obscure, dark, confused, uncanny world.

My head for a long time felt inside as if it were wrapped in a veil, a fog, so indistinct. All these days so unpleasant. . . . Later in the evening I felt so hurried inside, such rapidity, such unrest, and I did feel so weak. My head felt as if something had

turned round in it, I did not feel well, I felt as if benumbed, I felt as if something had turned in my head. . . . I am as half asleep, in a dream, but not a pleasant one; as if benumbed, it is not well with me, as if an attack were in me. . . . I ate something at mid-day, but a pity for the good things, I tasted nothing. . . . I do not feel at home yet in this house, though we have been here a long time. I seem, indeed, strange altogether, not only in my home, but in my life also. As if I were only half existing, that is long ago, not only the last weeks. . . . I can only express myself with great difficulty: it is hard work for me to talk. This afternoon my head feels as if everything in the right half of the head were as if changed, turned, transformed, up and down, upside down also in the right half of the head. . . . A short time in the afternoon I was not so deeply in this as-if-only-in-a-half- or hardly-at-all-consciousness in which I usually am. And I could rejoice, because our uncles spent a little time with us. But at once something began that became horrible. In the afternoon I had such agitation, such currents, no perception, and I felt so awfully bad at the same time and so on, and so often I had it in the left side of my head. And as I just then had to go to stool I felt so dreadfully ill, so miserable that I cried, called loudly because I thought that I was staying in a terrible attack; I cannot write down what horrors occurred to me later in the afternoon, during the stool which I had quite easily, which was only liquid. And where I had such awful pains in the left side in the head, it was something gigantic, stupefying me, I have never had anything like it. I am so terrified, so miserable, I must weep over myself in my despair. . . .

Saturday. It is something awful; I do not know what and where to, I am telling nobody anything of the terror that is with me from early morning, and, indeed, in the right half of my head all day long. I have been obliged to cry so much in secret; right in my head it feels awful, as if a meat-chopping machine were going and if everything in my head had been chopped into pap. And this chopping still goes on. . . . Why I can no longer write what actually is, I cannot, because I do not know how to, it cannot be described. . . . In addition, I am dizzy, giddy, in half or weak consciousness. . . . Since I opened my eyes early this morning there is something awful in my head. I don't know what is the matter with my head, it is horrible, something hindering my whole mental I, my thoughts, comprehension, hearing, seeing as usual. My head is miserable

to-day. . . . My head was too full, I did not even know what happened to me. It was so horrible in my head that I lay down on my back, thinking that it has now come to an end, now all the torture and agony will leave off. . . . I had to hold myself tight, so that all the confusion, these feverish phantasies, all the nonsense should not burst out. . . . It rustled and flowed uninterruptedly in my head; it flickers to and fro before my eyes. . . . I was not I. I still believe that death will come to release me. . . . I am not quite clear in my head, it is sometimes dull, and, as I must exert myself sometimes to understand what is said to me, it seems to me that I am an idiot.

On looking through the above sheets chosen from the diary, painfully kept by the patient day by day, it strikes one that there can be no doubt that the symptoms, as described one by one by the patient, correspond to the descriptions of dreamy mental states, as learned from Hughlings Jackson. It is also no incidental coincidence that in this patient again we find the loss of smell, as this symptom now plays a part anatomically explained by H. Jackson in the picture of dreamy mental states.

Moreover, it is worthy of remark that the patient reported the occurrence of various paræsthesias on the left half of the body, and that in separate convulsions a predominence was observable in the left half of the body. These are both conditions which point to a particular participation of the right cerebral hemisphere; this agrees with the observations of Crichton Browne (The Cavendish Lecture, "On Dreamy Mental States," 1895, p. 21), who notes in these cases—all right-handed persons—indications by modifications of sensation and movement in the left arm, that along with dreamy states there was or had been damage or disorder of the right hemisphere of the brain. The same author points out that Hughlings Jackson had long ago suggested that in epilepsy with the dreamy state the first spasm or abnormal condition would be observed in the left side of the body corresponding with involvement of the right or more subjective of the two hemispheres of the brain.

What I wish to lay particular stress on in the foregoing observation is the proof furnished by the entries in the diary

as to how the dreamy states, which at first only occurred in the form of attacks in our patient, assumed the character of a chronic course, and as a consequence, the painful nature of these conditions, which in this patient became almost continuously permanent.

It is a familiar experience with epileptics that when the convulsive attacks are omitted, either spontaneously or through the agency of medication, a condition ensues which is described by the patients as being so torturing that they would give the world to go back to former times when they suffered from the convulsive attacks. What is hitherto known about the subject and what has hitherto been described as to the condition can be thus summarised: That it is especially the morose moods and excitability which render such patients a veritable burden to themselves and their *entourage*. In the preceding observation we see the representation of an impressive mental picture, which shows how unbearable the non-paroxysmal time had become to our patient, owing to a dreamy mental state that had undoubtedly become chronic.

I had the opportunity, as Director of the Lunatic Asylum in Dobran, after the year 1880, of observing:—

Case 2 which I shall report more briefly: It refers to an epileptic whose attacks seemingly date back to previous syphilis and who had formerly suffered from typical epilepsy, from which, however, he had been nearly free for many years; after intervals, frequently of many months, there is a short spasmodic attack, mostly only occurring at night. There are, however, almost permanent conditions of a dreamy mental state that worry the patient terribly and have rendered him quite incapable of work. In the occasional conversations held with him there are always vague statements as to dreamy conditions, hebetude, decrease of consciousness, the most terrifying thoughts, loss of personal sensibility, all, however, accompanied by the constant assertion that he could not well say what really was happening to him.

In illustration of these statements I am giving a few extracts from the patient's diary, regularly kept for years.

Silence! Night is coming on—Body crumbles—Spirit withdraws—Soul—Departs! Tragedy ends.

Epilepsy always before my eyes. I dread and fear awakening out of the long stupefied condition. Understanding often stops. . . . I cannot pull myself together as to what is the matter

with me or going to happen; I then seek to dispel the obscure pictures by work, reading, praying, smoking, going into the open air, &c. This sometimes succeeds, thank God, but then the condition returns.

Dreams throughout the nights, then, on waking, day by day the terror of living with these tortures. The paralysis (spasm) in the left arm progresses. Unconsciousness (cannot remember) of my being assumes alarming dimensions. Cannot even remember anything of my marriage and the conduct of my business. I rise stupefied without regaining full consciousness, breakfast well, for my senses have not entirely left me. I pray, smoke, write, work to dispel the sad mood (half dream, half waking)—spirit leaves me, understanding halts—dreaming? stupefied? without memory? to bed.

Wednesday. Open my eyes, think it is 1 o'clock in the morning, instead of half past seven. As always, I am agonised because I am alive and have no mental power for life. It seems to me I have dreamt much. I remember something dimly.

November 1. The mental agony, I have gone through, the stings of conscience, the grief that I have forgotten the whole course of my life, only something passes before me like a mist, a shadow-picture in my mind that everything and that deep repentance have seized me so often, so severely, that a catastrophe has occurred. This time the fight was so hard, that I even believe it was a last one, that the Almighty decreed that the catastrophe was inevitable and worse than ever.

December 22. I awake at 6 a.m. and think it is night. Dreams; hunger overcomes me, senses vanish, I spring from the bed in despair, fear to lose my senses. . . . Am in a half-conscious condition, always in terror that I shall not come to myself or that *something* (imbecility? insanity? fit?) may come. . . . Soul fights as never before, think it is all over, beg somebody to fetch my near relations. . . .

December 24. I was awake from 2 to 3 a.m. Eat fruit, awoke before 7, remember, want to sleep on, drink cocoa at 8 o'clock. Then Oh! Almighty, catastrophe. In terror lest paroxysm occur; it does come. Senses commence to be confused, terror, fear, see the infernal regions, wish to take leave, but fear that I must wake again from death, hardly struggle, must not run away, cold sweat under my arms, arm already commences to be dislocated as in epilepsy, I groan, rub with vinegar, brandy. I believe that the good God has given me the power to stave off attack in consequence of my terrible daily *torments*. . . .

Tuesday. Agony, stupor, solace, have dreamt much. Want to sleep. Cannot wake, cannot sleep, about 10. . . . visited me. Can only talk to him in sadness. Get up at half past four, want to pray, sweat of anguish, terrible mechanical repetition, the mercy of the Almighty is incomprehensible.

I. I am often near to losing my senses.

II. Convulsions commence to attack me. (*No actual attack! Author's note.*)

III. Terror of death.

IV. Terror of re-awakening from death, and again other terrible presentiments.

And again, again, again thousands of times, the Almighty has called me back to life! . . .

Sunday. Can take no more notes. Misty condition becomes worse. I pray in agony. . . . My senses become confused. I pray to the Almighty to have pity on me for the sake of the poor children. . . .

Tuesday. Many dreams, awake at about 4; as always wish to remember, only atoms, fog-pictures of everything. Doze, dream on, wake at 6, dream, do not know again how to get my senses back, try to think how to begin, my spirit cannot go back, cannot go forwards! Exhausted. . . . am miserable. Have not the strength to describe my condition to anybody; and then nobody could imagine that such a thing is possible.

For years made notes for Prof. Arnold Pick, can never go to him; am afraid if I had to explain to him should get stuck.

Although the patient whose written statements are here reproduced has had considerably less education than Case 1, and some of his descriptions lack the delineative power that places her condition so strikingly before our eyes, they are able, nevertheless, to confirm the opinion of almost permanently lasting dreamy states.

I therefore believe that I have, by means of this communication, demonstrated that the condition hitherto only known to set in paroxysmally may also occur chronically in epileptics, incidentally rendering the period free from attacks excessively agonising.

I should like to direct attention to one more circumstance: It has struck me that these two patients, in contradiction to other epileptics who, though keeping diaries, only record their attacks in them, have confided to their diaries

for years an exhaustive description of the condition of their minds. Without attaching particular weight to the circumstance, I should like to point out an analogy, which is, perhaps, not quite accidental, with the typical neurasthenic patient who, on entering the consulting room of the physician, takes his diary or a long list containing an exhaustive description of his complaints out of his pocket.

In conclusion, I may remark that the bromides and apparently all other drugs have proved inefficacious in the conditions described above once they have assumed this chronic extension.

II.—On the Pathologically Protracted Duration of Impressions on the Senses as a Cause of various Disturbances of the Sensory Perception and especially of the Sight.

When recently[1] describing the "Bekanntheitsgefühl" of German authors, I mentioned the fact that neurotic and mental patients frequently reported alterations in this sense without any disturbance in the sensory perception being demonstrable. As it is a matter of course that it would be necessary to prove the *absence* of all or any such disturbances in order to be in a position to speak decisively of a change in the "Bekanntheitsgefühl," I laid no particular stress on the point.

It is very difficult to comply with this condition in each separate case, though at the first glance it appears very simple. We hear frequent statements of patients that this or that appears to them to be peculiarly changed, yet they are unable to say in what the alteration consists.

Bearing in mind the fact that in spite of the progress that has been made, our usual clinical methods of examination are only able to demonstrate the grossest disturbances; moreover, that physiology has taught us that every process of sensory perception consists of a series of complicated separate processes with exceedingly variable conditions, we must concede that there may be a disturbance of sensory

[1] *Neurol. Centralbl.*, 1903, No. 1.

perception in cases in which the disorder is situated in one of these separate processes.

In continuation of these studies I am anxious to draw attention to one such factor, which, though hitherto it has not been taken into consideration, cannot escape notice, when there is serious disturbance of perception; in cases less severe, however, it might easily give rise to the illusion mentioned above.

Every observer who notes the complaints of neurasthenics in regard to their disturbance of vision, must acknowledge that we entirely lack complete knowledge of the symptoms the patients complain of but cannot accurately describe, and that therefore we must by means of the teachings of psycho-physiology and psycho-physics strive more and more to learn to understand and analyse the single components of the "apparently psychical basis of a series of paræsthesias as, for instance, those in the region of sight."

V. Hösslin, whose words I here quote,[1] gives a series of such symptoms: "A few neurasthenic patients complain that everything appears to them in another light; that everything appears obscure to them, or that everything looks irridescent and glittering; that all objects make an altered impression on them; that landscapes or the expression on the countenance of people do not appear to them in their natural representation. These all are symptoms that are attributable, not to the altered function of the eye itself, but to altered perception in the brain."

The most recent nerve pathology, above all the wide borderland between cerebral pathology on the one hand, and sensory physiology and pathology on the other hand, affords an opportunity of understanding these and similar symptoms familiar to every nerve specialist.

Psychopathology has reaped the greatest benefits from this knowledge, and a series of disturbances of the sensory perception, such as are reported by patients, principally at the commencement of insanity, have become comprehensible to us. The deeper, however, we penetrate into this

[1] "Müller's Handbuch der Neurasthenie," 1893, p. 115.

obscure subject, the more we are impressed with the necessity for studying the separate factors that are set up in the sensory impressions, and these lines are given up to such a factor.

The observation I am about to report relates to a boy 14 years of age, whose family history was only slightly unfavourable. The lad was an excellent pupil in the gymnasium, and had always been in good health until he fell off a chair on the back of his head; according to all accounts he then exhibited the symptoms of slight concussion of the brain, and was therefore kept in bed for several days. When allowed to get up, he had no headaches, but could not walk properly, because he felt as if something were pressing on his head and limbs; when he went out for the first time, it appeared to him "as if the houses rose up from the ground." He since then had a bad appetite, heaviness of the head, especially on the left side, and he suffered from constipation. At the end of a fortnight he returned to school, but still complained that objects appeared peculiarly indistinct to him, as for instance, houses, especially when he looked at them from only a short distance. At this time one of his schoolfellows gave him a blow on the head with his school books, and his condition again deteriorated; he had the same symptoms as before; in addition everything seemed peculiar to him, and he had a sensation of giddiness, but not that of vertigo; on stooping he experienced a sense of heat in the head, he felt as if his head shook, when turning round he felt dizzy; he was also very timid and complained of occasional twitchings on different parts of the body; if he coughed he had pains in the top of his head, and he could not apply himself to his work. After his previous illness his eyes had been examined by an oculist, who found the condition of the eyes both functional and anatomical normal; on being now again examined the condition was the same. The boy again improved for a short time, and then had a relapse from a shock, a fire having broken out in the house. The examination of the patient, undertaken by myself, yielded the same negative result, as it did also in regard to the condition of

the nerves, and the only observable symptom was the weakness of the knee-jerk. During the examination the patient repeated all the complaints communicated above, which in face of the other condition, could only be regarded as the expression of traumatic neurasthenia. The peculiar disturbances of the sensory perception complained of by the boy remained incomprehensible, and at first were relegated to the category of obscure disturbances so often spoken of by nerve patients as discussed above, until on more narrowly questioning the patient, he made two statements, which, I think, assist in elucidating at least a part of these disorders.

The patient stated that when somebody passes him or when a vehicle drives past, he sometimes *still sees a shimmer afterwards*, and he completed this statement by observing that in addition he had a sensation *as if the object in question*, i.e., *the passing vehicle or person, were broader than normal*. *En passant*, I wish to call attention to the circumstance that this statement was made *quite spontaneously*, that it was also partially embodied in a written description of his sufferings, drawn up by the patient, and that it was not the result of any examination made in that direction, representing as it does the observation of an entirely new and unknown phenomenon. According to my opinion there can be no doubt that this symptom, taken in conjunction with a part of the disturbances, that could not be described by the patient personally, is palpably a disturbance of the well-known protracted duration of the impressions of light, which are also termed "after-images." (Nevertheless, in this case it is clearly differentiated from all other "after-images.") As is known, analogies can be found in physiological psychology, and, without going into the matter, I refer the reader to *Wundt* (Grundzüge der physiologischen Psychologie, ii., 4th edition, p. 162) in which there is an experimental arrangement quoted from *Plateau*, in which a certain manner of turning permits of an enlargement of the human countenance being observed, while a turn in the opposite direction shows it as if diminished in size.

I have now come to the conclusion that many other complaints of patients as to their sensory impressions might be

elucidated on the basis of this case. Thus, as to the vision in general, protracted after-images are well-known disturbances that affect the view of moving objects. The patient, however, related that, in regarding houses, the same effect was produced, thus not-moving objects stared at or regarded steadily may assume an abnormal appearance. This peculiarity is explained by the fact that our eyes, except in physiological fixing tests, are, as a rule, always on the move, or the pauses between each movement of the eyes only excessively short. It is therefore quite comprehensible that the after-image of impressions of vision, when pathologically changed, must also to a great degree affect the view of not-moving objects in so far as the after-images of the objects, seen in the pauses between the movements of the eyes, must, as it were, obtrude on those seen while the eyes are in movement.[1]

We are, moreover, aware that the duration of consecutive visual impressions plays an important rôle in the continuity of the act of sight, and it can therefore be understood that the disturbance of the after-image, as described above, induces a disturbance of continuity.

Even the disturbances of his hearing, as reported by the patient, might be included in the explanation hereby attempted. As a matter of course, the opinion formed of this complaint put forward by the patient might be that it was the hyperæsthesia of the organ of hearing, which is so common in neurasthenic persons, but this would not be quite justifiable in this case as an explanation for the existence of the phenomena. Just the complaint "everything echoes louder" makes it probable that in the acoustic region also the same disturbances are in question as those which we regard as the basis of the optical disorders. If then we assume that every acoustic sensation of our patient exhibits an increased after-sound (Nachdauer), it explains

[1] I should like only to assume that the peculiar indistinct impression produced on the pupil by optical impressions, as reported by him, may have originated in the same manner as the symptom described by Szili (*Zeitsch. f. Psych.*, 3, p. 371) as "fluttering." The author is of the opinion that the excitement, subjectively originated, survives the irritation and produces a powerful after-image; however, in this regard, the question does not appear to have been as yet solved in so far as to which plays the principal part, the positive or negative after-image (Compare Holden, *Archiv. für Augenheilkunde*, 1899).

this unpleasant increased "echo" by the fact that the after-sound of the sensation of tone plays as important a part in the physiology of hearing as the after-image does in the physiology of vision.

The facts belonging to this part of physiological psychology are, however, of more particular interest from the circumstance that they tend to the comprehension of some few other symptoms of our patient.

Experiments made as to the effect of the after-images show that, under certain circumstances, they give rise to apparent movements. I am of opinion that, with all reserve, this may partly be brought into connection with the circumstance that our patient reported symptoms of giddiness, which, as far as could be confirmed by examination, had nothing in common with either ordinary giddiness or vertigo.

It can hardly be positively asserted in which part the disturbances here noted should be placed, as even the same question as to the after-image in a physiological sense has not yet been decisively answered. The fact that the symptoms had arisen immediately after a blow on the occiput may lead to the assumption that the location of the morbid processes may be in the occipital lobes; nevertheless, there is no reason that this process may not also be located in the subcortical optical centres or even in the retina itself, in the sense of defective "retinal reparation" (Netzhauterholung), as we may conjecture that a disturbance affecting the optical centres in the occipital lobes may have some sort of influence on the last-mentioned region of the organ of sight.

This opinion is by no means in opposition to the view we have expressed in explanation of the hyperæsthesia of sound complained of by the patient, this being the result of the protraction of the sensations of hearing, for the concussion of the brain that preceded the illness may have equally affected the temporal lobes as the occipital lobes. Yet the assumption also of a peripheral cause for the symptoms coincides equally well with the etiology as the one in regard to the optical view.

I believe that my efforts here to partly explain the obscure disturbances of sensory sensations apply equally, not only to

neurosis, *par excellence*, neurasthenia, but that it also throws light on other symptoms equally obscure within compass of insanity without organic changes.

The transition from the symptoms discussed above to the pathology of psychoses seems given by different observations concerning patients with focal diseases of the brain. Thus Wilbrand reports ("Die Seelenblindheit als Herderscheinung," 1887, p. 587) in a patient suffering from bilateral partial hemianopsia: "The sight of her open wardrobe and linen-press throws her into confusion; she certainly distinctly sees the separate articles in her cupboard, but is always obliged to call her maid to her assistance when she wishes to find anything. This confusion is also originated when walking in the street, on seeing many persons, and is, in fact, induced by the many different retinal impressions affecting the eye unequally. She always descends the stairs backwards as the sight of the many steps makes her confused and giddy." One may surely be justified in explaining a great part of the symptoms reported in this quotation with the assistance of the recent knowledge on localisation in the optical region, the disorders of which, especially in regard to cases of bilateral hemianopsia, have been minutely studied. Nevertheless, I am of opinion that some of the disturbances reported by the patient, above all the symptoms caused by going downstairs, would be better explained by a protracted duration of the optical impressions.[1]

I find the proof for the correctness of this indication in another self-observation of the same patient, which she terms "perverted thought" (Wilbrand, p. 59), and in which the illusion is originated and temporarily maintained that the street is next to her sitting-room instead of her bedroom being there. Thus, when she has been rummaging in the chest of drawers in her bedroom, has locked it up, and returned to her usual seat at the window of her sitting-room, she is dominated by the impression that her chest of drawers is really standing in the street, and it was useless to have locked it, because it was still in the street.

[1] Wilbrand (p. 130) gives another explanation, but I consider the one furnished above in the text for a part of the symptoms to be decidedly more correct.

I regard this imagination to be the result of protracted duration of the perception, certainly increased to an unmistakeable after-image, and I also regard the condition as reversed, *i.e.*, that the protracted sensation is primary, not the illusion; that the "perverted thought" was a functional disorder, or only an indirect passing effect is proved by the subsequent cessation of the idea.[1]

In a case of agraphia published by Sérieux (*Compt. r. et Mem. de la Soc. de biol.*, 1891, Dec. 5) disturbances of vision of the patient are reported, which were manifested, for instance, in that she observed the shape of her daughter as "flat as a ray-fish" (*raia*). She said to the members of her family: "I do not recognise you"; "Your nose is altered"; "Your tongue is hanging out." At a later stage of the disease it was stated that she saw that her relatives had fantastic heads; she also had similar manifestations in regard to the doctors, to whom she smilingly observed: "They are like masks, the nose is changed, it is so comical." In this case there is no doubt that it was a question of symptoms, which one might classify under the general term psychic blindness. But if one deeply analyses the separate symptoms, a few of the illusions of the patient seem to indicate that the disturbances previously discussed play their part here. I have succeeded in finding another observation in the literature which may also probably belong to our category; Morel (*Du delire emotif. Extr. des Archiv. gén. de Med.*, 1866, p. 431), reports the optical hallucinations, or rather illusions of a patient: "The features of individual countenances appeared transformed *similarly to those india-rubber faces which can be drawn lengthwise or broadened at will.* It seemed to the patient that persons entering her room were making grimaces." The patient's description, which is in italics, seems to indicate that the pathologically protracted duration of the optical impressions gave rise to the grimaces by an apparent elongation of the faces. The disturbance here described partly simulates so-called meta-

[1] Willbrand, p. 142, explains the phenomenon by disassociation in the ordinary series of the reproduced optical illusions; the explanation given in the text appears to me to be simpler.

morphopsia, the symptoms of which may, perhaps, in part be attributable to the disturbances discussed above.

Epileptics occasionally state that they see everything vaguely, with undulatory motion, or as if through a veil; the assertions of insane patients who complain of giddiness may be drawn into the question. On narrowly questioning them, one finds that they suffer from nothing approaching actual giddiness, but only have the sensation of a certain uncertainty of their surroundings; the sensation of "flickering"; such flickering may originate from a sensory protraction, as in the physiological experiment, if the optical stimulations do not follow each other with sufficient rapidity.

No more instances need be quoted to prove that the study of the illusions may benefit from our study, only be it said that Mauthner (*Vorlesungen aus dem Gesammtgebiete der Augenheilkunde*, 1881, part 6-8, p. 532) directs attention to the fact that "so-called illusions may certainly frequently rest on a disturbance of the vision. By reason that the insane does not see objects distinctly he considers them to be something different. This would not happen if his powers of perception were normal."

The above observation has now demonstrated how factors in pathology, hitherto hardly noticed, may essentially influence the sharpness of the sensory perceptions, and the conclusion will be formulated that this factor, when accorded more attention to, will be found incidentally to represent disturbances.

The illusions reported by different patients as to the altered conditions of the size of objects seen by them (macropsia and micropsia) are certainly for the most part to be explained by disturbances in accommodation; perhaps, also, by disturbances in the localisation of space, but the subject is worthy of consideration and further research, whether at least in some cases the symptom here treated had exercised some effect.

An examination of all the literature accessible to me has done little to further my knowledge on the subject; Parish ("Über die Trugwahrnehmungen," 1894, p. 7), and before him Sully, ("Die Illusionen," 1884, p. 53) discusses under the head of hallucinations in consequence of incomplete and

pathological function of our organism, the illusions that take place in consequence of the after-effect of a stimulation, in consequence of the resonance of sensation continued for some time, and the impossibility of an exact separation of two successive stimuli. In conclusion, I refer the reader to a note by v. Kries (*Zeitschrift für Psychologie*, xii., p. 100) with which I have only recently become acquainted :—

"In another place, to which I refer you, I communicated the observation that to a person suffering from hæmeralopia it is impossible by any means to see the after-image. Further research on individual differences and *pathological modifications of the symptom* would be very interesting."

The conditions under which I saw the patient did not, as a matter of course, permit of a professional examination of the disorders in the higher sense. I believe, however, that I have accomplished my purpose if I have succeeded in directing the attention of my professional colleagues to pertinent cases, and given the impetus to apply suitable methods of examination, and thus perhaps open a fresh path in the region of disturbances of sensory perception, which, in spite of all, are still obscure. Supplementarily, I find something on this subject by Ireland ("The Blot upon the Brain," 1885, p. 195). The author places uncontrollable impulses in analogy with the sensory after-image; after having mentioned the physiological appearance of the after-images, he expresses himself as follows: "But with some people in unhealthy conditions such sensations are more persistent. If one pinches or pricks them they do not feel very quickly, but the painful impression remains. The perception of colour persists for some time, so as to hinder the perception of a new colour; on looking at a face or a row of faces, the images remain for some time after we have withdrawn our gaze from them."

III.—On Reduplicative Paramnesia.

I recently published an account of "a new form of paramnesia" in the *Jahrbüch für Psychiatrie und Neurologie*, vol. xx., which relates to the observation of a

paralytic, and points out similar symptoms in delirium tremens, &c.[1] I am now in the position to supplement this communication with another, which not only confirms the first one in a satisfactory manner, but also furnishes some further elucidation to the obscure psychology of the phenomenon.

In addition, however, I consider I am justified in my resolution to give this form a particular name, and as an antithesis to the forms of paramnesia hitherto known, and also so as to bring the particular characteristics forward, I have chosen the term "reduplicative paramnesia." This symptom, namely, is characterised[2] in that a *continuous* series of events in the patient's remembrance, subsequently fall into manifold occurrences; the isolated events, though they remain pretty clearly in his memory, are impressed on him as repetitions thereof. As an example: The patient in question asserted that there were two clinics exactly alike in which he had been, two professors of the same name were at the head of these clinics, &c. In this case weight was laid on the circumstance that there were *identical* groups of memories. A condition somewhat similar was manifested in a patient who has been in the clinic for a considerable period suffering from dementia senilis.

The case relates to a woman of 67 years, who was admitted on January 4, 1903. Apparently without pathological heredity she had been normal till lately. During the last few months she had lost strength rapidly, and psychopathic symptoms had supervened. Depressed moods, with superficial auto-accusations; ideas that she had nothing to live for, as well as distinct signs of forgetfulness, from which emanated delusions that she was being robbed, which ideas were, however, as quickly forgotten; in addition, there were illusions of memory (*Erinnerungsfälschungen*), apparently dependent on nightly dreams, supported by a disorientation of time (*zeitliche Desorientirung*), which again tended to the formation of delusions, as, for instance, as to the object of the presence of pupils with her daughter, a teacher. An apparent fainting fit that occurred a short time previously appears to have been an attack of apoplexy.

[1] On a later opportunity (see *Neurologisches Centralblatt*, 1903, p. 5) I have mentioned something similar to the subject here discussed.

[2] See p. 22 of reprint.

On admission to the clinic the patient essentially exhibited the traits corresponding with the picture of presbyophrenia described and so named by Wernicke, and which appears already in the above sketch of anamnesis. Her power of memory is considerably diminished; the opinion in regard to surroundings is mainly correct, but is easily affected, if not entirely obliterated, by accessory circumstances. The mood is easily fluctuating, in general, however, serene; incidental mistaking of the surroundings; illusory hearing of any sounds easily originate delusionary misapprehensions, accompanied by lively confabulation, the products of which were often maintained in her memory for a long time. In the meantime the patient is able by her dignified demeanour and a certain wit to deceive one as to her mental defects.

The bodily condition conforms to the age of the patient; slight difference in the pupils, the left somewhat wide, and does not react to light; the nervous system otherwise normal. On May 5 she had a convulsive attack in the night, which, according to the statement of the attendant, affected all the limbs. The face was thereby drawn to one side. Next morning the patient was feverish, temperature 38·2, had diarrhœa and vomiting; during the whole day she lay quite apathetic, and passed fæces and urine under her; no sign of excitement. Next day she was better.

On May 20 she again lay in bed in a stupor without preliminary symptoms having been perceptible; she again had slight fever, and passed urine and fæces under her; she was highly sensitive to needle pricks; next day she was again better.

On May 24, during the morning visit, she enquired after her daughter, she imagined she was in K., and in reply to the assistant's question how it was that he was in K. also, she said she was very pleased to see him *here too*.

On being questioned further how it was that the entire hospital, as well as the patients, came to be in K., she replied that the doctor had so arranged it. When examined later on, she recognised the author, but at first does not know where she is; had at first believed that she was in K., her birthplace; everybody had said, "Now we start for K." She now conjectures there is something behind all this. On being asked how it was that the professor had come to K., how had the entire surroundings come there, and to the objection how could the doctors have come there? "Why, good God! everything can go round about and back again." When she is at first led to a sick room she does not recognise it, but when led into the room in which she had stayed last, she recognises it, but

explains that she has only occupied it since yesterday. Where had she then come from? From Prague; she does not know how to explain who brought her here; it had already happened once previously that she had been conveyed into a lunatic asylum by mistake she said. Being more closely pressed to say where, then, we are now, she says, in a suburb of Prague; on her attention being directed to her contradiction, she explains that her statement in regard to K. was a mistake, which had occurred by the above quoted assertions of those around her; during the further course of the cross-examination she becomes excited, it was all trickery, her clothes had been taken away, &c.

Is this a hospital? "You must know that!" How do I (*i.e.*, the Professor) come to be here? "That is not my business; if it were clear to me how it really is, I should have no respect for it." She then continues excitedly: there had been a great swindle, and she had been dragged into it; after being soothed, she relates that she has been in the clinic in Prague for five months; she had left there yesterday; this is a clinic, too, exactly like the one in Prague; her daughter had visited her yesterday (correct!) and had promised her a visit. She at once recognises two doctors that now enter, and to the question from where she knew them, she answers: "From your place" (*i.e.*, from the clinic in which she had previously been). Shortly afterwards, when again visited in her room and questioned as to the peculiarities of the two clinics, she says the "to-day's" clinic is perhaps a continuation of the previous one; at present she was in a suburb of Prague, which she mentions by name; in the first clinic she had also slept in an iron bed, but she does not know if it stood at the same place pointed out to her; she recognises a few of the patients as having occupied the same room as herself for a long time, and asked how they had managed to come here in the second clinic. She says: "They come from one place to the other." She again explains this is the same clinic as the former one, but at a different place. When later on her son pays her a visit she is still excited about her new place of residence; wishes to leave; she informs an attendant she was at the Professor's in the other clinic; in the evening she is still excited, asks to be spared further questioning as to where she is, and considers it ridiculous that the professor asked her about her bed.

On the following day she is somewhat distracted; is at first under the impression that she is in a private house, but later on comes back to yesterday's statements. Now she is in the "suburb clinic," in contradiction to the "town clinic," where she

was formerly for five months. The same professor attends at both clinics, and the assistant of the "town clinic" also comes here; specially interrogated by the author as to if there are two professors of the same name, she says, "he is the same"; and adds, smilingly, "you are everywhere."

Interrogated as to her acquaintance with different patients, she says, for instance, of one, she had known her from the *other* clinic; she also knows the assistant of the male division (whom she does not see so frequently) as from the "*old*" clinic. Asked about a patient who had only recently come to the clinic, she says she has only known her from here; a patient, on the other hand, who had been longer in the clinic, she knows from the "*town clinic*."

The same condition is still present on the 27th. The two clinics are quite alike, even the head attendants are quite alike. An utterance that she spontaneously made to the assistant at this time is characteristic: "This is already the second time that I have had the advantage of your valued treatment," During the next few hours the symptoms are manifested by numerous delusions of remembrance, alternating with misapprehension of the locality, but the illusion as to the two clinics has disappeared; she knows she is in Professor Pick's clinic, and knows nothing of a second clinic; it is possible there may be one, but she is unacquainted with it.

On May 28 an episode, similar to the one just described, again occurred. In the afternoon she had had a visit from her daughter; later on she fell asleep, and awoke at 6.30 p.m. She was under the impression it was morning, and asked for her breakfast. In the evening she tells of the shopping she has done in town, and then as follows: She had spoken to a young and beautiful patient, who was in the third storey (thus a storey presumably higher than the clinic itself). Up there it was exactly as here in the clinic; there was also room No. 118 (that is, the patient's room). The patient cannot by any means be induced to relinquish this idea, in fact, she laughs at the assistant's efforts, and says "he is making fun of her"; after a long argument that the clinic is in the second storey, and there is no storey above it, she says: "Then we are in the first storey, and that was on the second storey; it is so difficult for me to go upstairs, then the first storey appears to me as if it were the second."

On the following day she kept to her tale that she had been in the third storey and had there conversed with the young patient "it was there that I was *formerly*." Were you in a clinic there? "Oh yes, certainly, that was so." How long have you been here?

"Only about three or four days." The other clinic had looked exactly like this one. The head attendant there was also the same.

Being on the 30th inst. reminded of her statements in regard to the two clinics she said that the second one was outside in . . . (could not remember the name.) On June 2, she again made a statement regarding the second clinic, which was supposed to be a storey higher; she had been examined up there by the professor; when the assistant, to disabuse her, led her into the examination-room close by, she explained that it was certainly similar to the one she was usually examined in, but that one was situated a storey higher.

If we except the typical kind of confusion, caused by delusions of memory, the first as well as the above-mentioned further episodes represent that form of paramnesia which I have formerly described and preliminarily termed "reduplicative;" the similarity with the previously described case is the more striking as the phenomenon in both cases is connected with the memory images of their stay in the clinic; we here observe, also, that an incidental temporary interruption in the course of events may divide the connection thereof and in the one case this was probably induced by a short sleep; we observe, moreover, how, although the patient was aware of the similarity of the essential parts of the events before this interruption, with those after it, they nevertheless became duplicated in her mind.

The case here reported differs from the case formerly reported inasmuch as, our (female) patient did not, like the first patient (a male), duplicate the professor and the assistants, but that she asserted something similar in regard to the arrangements of the clinic and *a number* of the patients, but she had no doubts as to the identity of the author as the administrative head of *both* clinics, nor of the assistants being the doctors acting in *both* clinics. It is not difficult to give a right explanation for this difference. When discussing the first case I expressed myself as follows: "Supposing that somebody else, even a person as mentally weak as our patient, but without his disturbance had gone through the clinical stay, the stages of which he later on

regarded as different events, such a disassociation would certainly not have set in because the number of mental impressions, identical in both periods of time, and the corresponding and connective pictures of memory would not have permitted such a disorder of continuity to occur. It is different with our patient. Undoubtedly this continuity did not, in its entity, come to his consciousness; the influence of alternating events, which for every other person would have been of very little importance, became predominant, and thus the uninterrupted series of events are to him, divided into two different groups." Our female patient is perceptibly different from the paralytic, by the fact that she has retained much more intelligence than he; this is especially manifested by the fact that in the course of conversation she tries to give an *explanation* for this or that remarkable circumstance, and that she therefore also does not accept the attempts of the doctors to deceive her.

That the greater intensity of certain memories also plays a part in this is demonstrated by her statements in regard to the identical patients being present in the two presumptive clinics. In the first case (see reprint, p. 26) I called attention to the fact that the stronger memories, inducing the continuity of occurrences, only went far enough to convey to the consciousness of the patient a certain similarity between the apparently double clinics and other occurrences; in our new case we observe how a higher degree of intelligence only permits a part of the stores of memory to be duplicated, whereas the identity of important single occurrences is firmly retained by the patient. We may also observe an essential difference between the intellectual disturbance of our patient and that of the paralytic, by which we have sought to explain the paramnesia of the latter (see reprint, p. 29).

Our patient is still further differentiated from the first case by the circumstance that already a short time subsequent to the apparent double experience she does not presumably know how to recount it, but evidently does not wish to do so, whereas the paralytic for a long time after repeats the story of the two clinics, two Professors Pick, always in the same way, monotonously and without judgment. This is certainly partly due to the diminished retaining power of

memory of the patient, corresponding with her senium; partly, also, to the better power of judgment of the female patient, which leads her to doubt the reality of these double events.

In the work repeatedly quoted, I had directed no attention to the question of the "Bekanntheitsgefühl," and I only introduced it into psychiatry as a cause of other symptoms (see *Neurologisches Centralblatt*, 1903, No. 1), and it was only in this last paper that I connected the peculiar paramnesia therewith. I would now suggest that this question should be brought under discussion. Volkelt (*Zeitschrift für Philos. und Philos. Kritik*, 1901), after Hoffding had considered the same phenomenon as "sense of familiarity" (Bekanntheitsqualität), has pointed out that this sense which he thus terms plays an important role in the remembrance, especially in recognition, and that which he terms "certainty of memory" is the chief factor which qualifies his own experiences as memories. I was formerly of opinion (see reprint, p. 5) that the phenomenon of reduplicative paramnesia here discussed originated mainly from a disturbance of the "sense of familiarity," that when this failed the situation just experienced would not be a repetition of the last, but a new impression. At present, after repeatedly thinking over the new observation, I have come to the opinion that the affair is somewhat different. The patients I have reported had "sense of familiarity" as well as Volkelt's "certainty of memory," and in consequence there appear to them, on one hand, the pictures of memory, as if like the situation just in its course; on the other hand, they are sure that they have actually experienced the earlier situation; but, in consequence of mental disturbances (in the first patient through paralytic dementia, in our patient through dementia senilis or presbyophrenia) they cannot identify one situation from the other, which become duplicated and eventually divided into a multiplicity of events.

In conclusion, it must be remembered that, like in the former one, the case discussed, though nearly related to cases termed "double or alternating consciousness," had not yet reached that stage in consequence of the maintenance of the consciousness of personality.

Reviews.

Archives of Neurology, from the Pathological Laboratory of the London County Asylums, Claybury, Essex. Edited by FREDERICK WALKER MOTT, F.R.S., M.D., F.R.C.P., Director of the Laboratory and Pathologist to the London County Asylums. Vol. ii., 862 pp. (London: Macmillan & Co. 1903.) 25 plates and numerous figures in the text.

IN this the second instalment of researches carried out in the laboratory of the London County Asylums Board, the high standard of excellence, of which we obtained a foretaste in the first volume, is more than maintained, and the production in general reflects the highest credit alike on the editor and his collaborators, on the publishers, and, above all, on the members of the Board, who from the first freely recognised the value of scientific investigation, and are already reaping the fruits yielded by an intelligent elucidation of some of the many problems bearing on insanity.

The subjects dealt with in the various articles cover such an extensive field that it will be necessary to discuss each separately.

Tabes in Hospital and Asylum Practice. By F. W. MOTT, M.D., F.R.S. 327 pp.

In the dual capacity of physician-in-charge of out-patients at Charing Cross Hospital and pathologist to the London County Asylums, Dr. Mott has been favoured with facilities, denied to most men, of observing not only the ordinary cases of tabes which hospital physicians are familiar with, but also those in which the onset of mental symptoms necessitates confinement in asylums; and as a result of several years' patient study we have now laid before us a monograph which we consider one of the most elaborate ever written on the subject.

The basis of the article comprises records of thirty autopsies (in the majority of which a microscopic examination of the brain, spinal cord, ganglia, peripheral nerves, optic nerves, retinæ, &c., was carried out), clinical notes of sixty cases of tabes dorsalis, and of the same number of the tabetic form of general paralysis, and lastly, an account of the sensory disturbances in no less than fifty instances, and in regard to the latter the author justly observes that the amount of work involved in it can only be appreciated by those who have had a similar experience.

In an etiological survey decided statements concerning the identity of tabes and general paralysis are made, for instance, "etiologically and pathogenetically there is one tabes which may begin in the brain (especially in certain regions), or in the spinal cord in certain regions, or in the peripheral nervous structures connected with vision, or in nervous structures connected with the viscera, constituting, therefore, different types, any of which may be present or be associated with one or all of the others."

Well-known as one of the champions in this country of the view of the syphilitic genesis of general paralysis, Dr. Mott necessarily takes a similar stand concerning the origin of tabes, and, in fact, brings reliable evidence of syphilis in 75 per cent. of the cases in this series. Strengthening this point some interesting figures are given showing the high proportion of deaths in infancy of the children of tabetic and tabo-paralytic subjects, the number of miscarriages, and the relative frequency of sterility in the female subject. Further, to the list of cases of conjugal tabes and general paralysis reported by Mendel and Raecke, cases which are extremely suggestive of the syphilitic origin of the diseases, seven more are now added. In addition to syphilis the action of other causative factors in the production of both tabes and general paralysis is duly recognised; thus the report of the case of the mounted policeman, who was first affected in the arm with which he controlled the reins, is a vivid example of the importance of stress; and as to hereditary predisposition, the figures show that this does not appear to be so important in tabes as in general paralysis.

The enquiry indicated that the average age at which ordinary tabes developed was 37, and that the interval between a definite history of infection and the onset of symptoms was usually fifteen years, the shortest period being four, and the longest twenty-six years. In 60 per cent. of these cases shooting pains constituted the initial symptom, in 14 per cent. visceral troubles, in 12·3 per cent. diplopia, and arthropathies and blindness each in 10 per cent.

Coming next to the symptomatology, we find first some telling observations on the importance of pupil rigidity, to the effect that if this exists, combined with any of the other symptoms or signs, a diagnosis is rendered possible, the reasons being that the only diseases in which such pupils exist are tabes, general paralysis, and acquired or congenital syphilis; and in this connection the author relates how an examination of thirty-two cases of cerebral tumour at Claybury proved entirely negative in all but one instance, and then the subject was a general paralytic.

The occurrence of optic atrophy receives special attention, and was found in 30 per cent. of the cases of hospital tabes, and in at least 35 per cent. of the asylum cases. And it is noted that its onset is to be regarded as a serious indication, because it is a frequent forerunner of the extension of the disease to the brain. The correctness of the law of Benedikt, however, that tabetic motor symptoms vanish the moment optic atrophy appears is denied. Of more importance is the observation that in 50 per cent. of cases optic atrophy is succeeded by tabo-paralysis.

The sensory disturbances have been particularly well worked out, and from a large accumulation of results we have selected the following. The frequent diversity of precise distribution of the anæsthesia, analgesia, hyperæsthesia, hyperalgesia, is to be explained by the variable degree of complication of the peripheral afferent nerves. The lancinating pains, variable in intensity, distribution, and duration, abate or cease in the third stage of the disease when the roots which are undergoing degeneration have become destroyed; these pains, in the case of tabo-paralytics, are often insanely interpreted. Paræsthesia of the arms affects the post-axial border of the limb, that is, the distribution of C. 8, D. 1, D. 2. Trunk anæsthesia to light tactile impressions is the earliest and most constant sensory disturbance, and such anæsthesia is present in all cases afflicted by gastric crises. The anæsthesia corresponds to zones of distribution of posterior nerve-roots, and the zones may contain bands of hypalgesia or analgesia, or scattered points of blunted painful sensation. The zone corresponding to the distribution of the fourth and fifth dorsal roots is that which is most commonly anæsthetic. In an upward direction the anæsthesia does not extend beyond the second intercostal space, the reason being that the upper cervical roots entering into the formation of the brachial plexus are rarely diseased. In thirty-three cases the legs were analgesic or hypalgesic. A zone of hyperæsthesia often borders a zone of anæsthesia, and such a zone is commonly localised above the

third and fourth interspaces or below the seventh and eighth; in such cases the hyperæsthesia is an indication of irritation of roots prior to destruction, and its special localisation is due to the fact that the roots at this level suffer later than the mid-dorsal and lumbo-sacral roots. The onset of mental symptoms has an important influence on the diminution of the anæsthesia and analgesia. Thermo-anæsthesia was not met with nearly as often as light tactile sensations.

Concerning visceral disturbances, it was noted that bladder troubles were among the earliest and most constant; also that gastric crises occurred in 36 per cent. of the cases, and rectal crises in 8 per cent.; but no instances of intestinal crises were met with, and laryngeal crises only twice, and then in an early stage of the disease. As might be expected, these crises often accounted for delusions in the insane cases.

Arthropathy and bone affections occurred in 10 per cent. of the cases, being commoner in females than males, and frequently associated with gastric crises. Several cases are quoted to show that hard occupation involving much use of the joints predisposes to disease; take, for instance, that of the stonemason, in whom a large painless swelling of the right shoulder-joint was attributable to the use of the hammer; in other cases a trivial injury, such as a knock, was the starting-point of the mischief. Bearing on this question some judicious remarks are introduced on the importance of keeping in mind the possibility of spontaneous fractures in asylum cases, lest unjust charges of ill-treatment should be levelled at officials and attendants.

Passing next to the indications of brain affection, it is pointed out that as in general paralysis, so in tabo-paralysis the onset of the cerebral symptoms may be sudden. Commonly a seizure, recognised as congestive, apoplectiform, epileptiform or migrainous, is the discharging factor. Although changes in character may have been noticed by the patient's wife, relatives, or associates long before, a variety of acute mania is the commonest mental disorder which brings the tabetic or tabo-paralytic to an asylum, and it is interesting to learn that while the symptoms of cord affection at this stage may be well defined, the brain may show only slight macroscopic but distinct microscopic changes. Hallucinations are to be considered the most important signs of mental disturbance, because they exercise a powerful influence, even more powerful than delusions, on the intellect and volition of the patient, and the fact is noted that visual hallucinations existed even in those cases which were blind

from optic atrophy. It is remarkable that in tabetics with associated insanity their pains and sufferings are frequently converted into delusions of persecution, whereas tabo-paralytics are dominated by delusions of grandeur, exaltation, and a general sense of *bien-être*, and it is also important to observe that the grandiose delusions are more transient than those of persecution. Finally, although dementia of a progressive kind exists in all cases of tabo-paralysis, death usually ensues before it reaches the advanced stage seen in ordinary cases of general paralysis.

Morbid Anatomy and Pathology.—The microscopical appearances in various parts of the nervous system are so thoroughly gone into that it is impossible to do them justice in a brief survey; however, in regard to the posterior roots, we may mention that while the changes in them are accurately described, it is disappointing to find that the possible pathogenic influence of a co-existent meningitis does not receive the attention it deserves, especially in view of recent publications. As to Marie's theory, based upon the neuron doctrine that the degeneration is due to the nutritional change in the posterior root ganglion, Dr. Mott is convinced, from an examination of many ganglia, that the changes in them are altogether too inadequate to meet with that view, and is accordingly in agreement with most other writers on this point. In twenty-eight spinal cords examined it was invariably noticed that the three sets of exogenous coarse fibres entering the cornu-radicular zone and proceeding respectively to the root zone of Charcot, to the cells of Clarke's column, and to form Goll's column were affected, not always, however, to the same extent. Degeneration of the first sacral root which supplies the sole of the foot and is so frequently involved in tabes, is alluded to as a potent factor in the production of ataxia of gait and station, because it means severance of guiding sensations to the cerebrum and cerebellum, and the statement is also made that the ataxy has a decided relationship to the atrophy of the plexus around the cells of Clarke's column, because no case of marked ataxy came under observation in which this change was not pronounced. In a few cases the fine fibres of Lissauer's tracts were not affected at all, and in cases where the sensory cutaneous disturbances were investigated during life, it was found that there was a relationship between the degree and extent of the same, and the degree and extent of the atrophy in Lissauer's tracts. As to endogenous systems of fibres in the posterior columns, the condition of these were investigated in nine cases in which the ataxic symptoms were very marked, the descending endogenous system

(comma of Schultze, oval area of Flechsig) was affected more or less in every case, the ascending endogenous system, (cornu-commissural of Westphal) was never completely destroyed throughout the cord. In these cases of endogenous system degeneration there was invariably a very marked atrophy of the exogenous system, and there was a decided relationship between the degree of affection of the endogenous systems and the degree of ataxy.

The cells of Clarke's column were degenerated in some advanced cases, and as a result atrophy and sclerosis appeared in the ventral and dorsal cerebellar tracts. But a far more important associated degeneration was found in the pyramidal systems of fibres occurring in twenty out of twenty-eight cases. This degeneration is looked upon as evidence of cortical affection and is due either to chronic atrophic changes in the cortical psycho-motor neurons or acute destructive changes. Two varieties of change are described, (1) a form which is slow in progress, demonstrable by methods for the display of sclerosis rather than the method of Marchi, and which is more marked in the lower parts of the cord than the upper and only involves the lateral tracts, sometimes unequally. The fact that the lower parts of the cord suffer more than the upper is ascribed to a slow progressive atrophy of the neurons with the longest axons and furthest remote from their seat of nutrition, but it would appear although the author does not mention it that the change is of the nature of a retrograde atrophy. (2) The second variety is demonstrable by the method of Marchi, it affects both anterior and lateral pyramidal tracts and is traceable from the cortex downwards. Clinically it is associated with epileptiform seizures, and if these attacks be unilateral the acute degeneration will correspond, and if the convulsions be general the degeneration will be bilateral. It is ascribed to the direct effect of destructive changes in the cortex, and the observation is made that where the pyramidal degeneration is more marked on one side than the other, there is invariably a greater wasting of the corresponding hemisphere.

Coming lastly to the changes in the brain, it is observed that in tabo-paralysis these do not differ essentially from those of ordinary general paralysis, except that, generally speaking, they are not so advanced, and are of a more chronic nature. Microscopic changes in the nerve-cells, nerve-fibres and neuroglia are not discussed here, because Dr. Watson deals with the subject further on, but some suggestive remarks are made concerning certain cells which in an acute severe case of general paralysis are seen in abundance on the vessel walls and in their surround-

ing lymphatics—cells which stain deeply with basophil dyes, which go by the name of plasma cells of Marscholko and which Vogt regards as pathognomonic of this disease. With the latter conclusion Dr. Mott is almost in agreement, because only in one case of general paralysis (a juvenile) did he fail to find these cells and in the brains of many other conditions examined they were only found in one case of Congo sickness and one of multiple syphilitic gummata. According to the author these bodies exist especially in those regions (fronto-central) of the brain which generally show wasting in general paralysis; he is also in favour of the conclusion that they are derived from lymphocytes and not from endothelial cells, and as firmly believes that their abundance is associated with the amount and the intensity of the acute neuronic irritation and destruction. Concerning the nature and source of the irritant in these cases Dr. Mott practically repeats his views expressed elsewhere that we have to deal with the formation in the blood of a toxine which has a special tendency to fix on to certain portions of the nervous system which either stress, heredity, or anatomical conditions place in a lowered state of resistance, and in spite of our lack of knowledge of the specific germ, he says that facts warrant the assumption that the irritant is a latent toxin of syphilis which has a special affinity for nervous structures, a toxin which can only operate under certain abnormal metabolic conditions of the neurons.

In closing this digest we must state that it has been impossible for us to give due notice to the accumulated mass of facts and masterly considerations which the monograph contains, and to those requiring further details, we would say that not only will the general remarks repay careful perusal, but there is not one of the seventy-four cases used for purpose of illustration that is not full of interest.

Amentia (Idiocy and Imbecility). By A. F. Tredgold, L.R.C.P. Lond., M.R.C.S.Eng. Late Technical Research Scholar of the London County Council. 94 pp.

Restricting the term amentia to those cases in which arrested development takes place before the age of 12 or 14 years, the effects of various influences upon the developing brain are considered in the following way: (*a*) intrinsic or hereditary influence on the germinal plasm; and (*b*) extrinsic or environal, these being, before birth, the mental and physical conditions of the

mother; at birth, the accidents of labour; and, after birth, the influences of trauma and disease.

Accurate and complete family histories in 150 cases were obtained and upon these the etiological and clinical survey is constructed. Regarding hereditary influences, the clinical histories show ancestral abnormality of the nervous system in 85·5 per cent.; but the conclusion is arrived at that "there are no grounds for thinking that one kind of mental disturbance more than another is liable in subsequent generations to be transformed into idiocy, also that the insanity in the parents need not necessarily have made its appearance before the birth of the patient." Syphilis in the progenitors was certain in 2·5 per cent., possible in 3 per cent. Hence, the author believes that this malady can only rarely be looked upon as an exciting cause of idiocy; in the presence of other adjuncts, however, it assumes a dangerous *rôle*, and he considers that its potency as a factor for evil is more often to be found in degenerative processes, *e.g.*, general paralysis, than in the production of developmental arrest. No great significance is attached to such conditions in the mother as mental worry, shock, or maternal impression, physical ill-health during gestation being held to be of more importance. Excluding cases complicated by a history of hereditary predisposition, it is considered that in only 1·5 per cent. idiocy may be attributed solely to the dangers of parturition: this small figure is contrasted with that (17·5) given by Beach and Shuttleworth. The author is inclined to minimise such factors as instrumental interference, protracted labour, and to a lesser extent asphyxia neonatorum, but certain portions of his remarks in this connection are neither particularly clear, nor free from ambiguity; also his statement that the artificial alteration in the shape of the infant cranium practised by some races has apparently no evil effects does not appear to us to assist his argument. Little importance is attached to so-called teething convulsions, the cases in which their occurrence was recorded having mostly a well-marked history of hereditary taint. We may also add that the differential diagnosis between an epileptic fit and a teething convulsion is not always made, and that epilepsy may be ascribed to so-called teething convulsions which were probably true epileptic fits. Trauma, if slight, is not believed to be of great potency unless in the presence of predisposing elements, but a greater degree of injury is undoubtedly an exciting cause.

The author makes out a strong case in favour of the statement that amentia is not a "freak of Nature," and, laying stress on the

toxic actions of alcoholism, tuberculosis, syphilis, &c., on the germinal plasm, and referring to the theories of Weismann, he asks how it can be conceded that such influences may have so marked and destructive an action upon the somatoplasm, and at the same time leave the germplasm unaffected. He argues that "idiocy is in most cases the final expression of a tendency to degeneracy, which, first manifesting itself in deficient inhibitory power and unstability of the nervous system, as is seen in hysteria and the like affections, passes, if unchecked, along its downward course, next revealing itself in epilepsy and various forms of insanity, to finally culminate in idiocy and the extinction of the family."

The last part of the paper gives the results of an examination of twelve cases—nine of primary amentia, two of secondary (or accidental) amentia, and the twelfth an epileptic without amentia —for purposes of comparison. In the primary aments it was found that the skull was thicker and denser than normal, in most cases the condition of the membranes varied with the amount of dementia that had supervened; in idiots the brain was small, the hemispheres sometimes markedly unequal, irregularly convoluted, and in some cases the seat of gross lesions. The nerve-cells showed, (1) numerical deficiency, especially of the small and medium-sized pyramidal cells; (2) irregularity in arrangement, in the pyramidal layers they were seen lying obliquely, horizontally, or even completely upside down; (3) imperfect development of individual cells, this being evidenced by alterations in the appearance and eccentricity of the nucleus, absence of processes, scanty protoplasm, &c., all suggestive of the embryonic condition. These appearances were most marked in the small pyramidal layer and the frontal and parietal regions. The presence of pigment was also noted as a very common feature. The fibres, stained by the Kulschitzky method, showed diminution, particularly in the outer line of Baillarger, next in the super- and inter-radiary systems, and less in the superficial tangential fibres. We note, however, that no remark is made as to the condition of the radiary bundles. Neuroglial changes were found; in some cases there was an enormous overgrowth (sclerotic amentia). Pigmentary deposits were found in the endothelial cells of the vessels. In the cases of secondary amentia (both epileptic) the microscopic appearances differed from the above, and appeared to be a cell-degeneration process supervening on an imperfectly-developed condition; the cells chiefly affected were the small and medium-sized pyramids, many of which contained black pigment.

The Histological Basis of Amentia or Dementia. By J. S. Bolton, B.Sc., M.D., B.S.Lond., Assistant Pathologist and Medical Officer in Charge of Patients at Claybury Hall. 197 pp.

This paper consists of two parts; the first deals with the morbid anatomy of dementia, and a number of observations on the general pathology of mental disease are added; the second is devoted to histology.

In Part I. the author compares the mental conditions with the naked-eye cerebral changes in 200 consecutive cases of insanity, in order to demonstrate that the morbid alterations existing inside the skull-cap in insanity vary in degree with the amount of dementia present, and are otherwise independent of the duration of the mental disease. Finally a series of types of dementia paralytica and the relationship of this disease to ordinary insanity are dealt with.

The 200 cases are arranged clinically into five groups, according to the amount of dementia observed, and these groups are further subdivided into a number of classes, so that finally an extremely elaborate and complex classification is arrived at. But since this classification depends on a finely-graded estimation of the degree of dementia present—an estimation into which the personal equation of the observer must largely enter—and since it has apparently been found impossible to subdivide the groups by any common factor—clinical, etiological, or otherwise—such an arrangement is necessarily highly artificial.

A table is given to show the relative amount of thickening or adhesion of the dura, subdural deposit, subdural excess of fluid, pia-arachnoid, subarachnoid excess, dilatation and granularity of the lateral and fourth ventricles, in the various classes; from which it is gathered that the morbid changes vary directly with the amount of dementia present.

The author finds that wasting of the cerebrum during the progress of dementia is most marked in the prefrontal region (anterior two-thirds of the first and second frontal convolutions, including the neighbouring mesial surface, and the anterior third of the third frontal convolution), next in the remainder of the first and second frontal convolutions (Broca's convolution being equally affected in cases of dementia paralytica), next in the ascending frontal and Broca's convolutions, next in the first temporal convolution, and superior and inferior parietal lobules, and lastly in the remainder of the cerebrum.

The relationship of degeneration of the cerebral arteries to mental disease is discussed, and the conclusion is arrived at that "in the cerebrum which has begun to break down, or where degeneration has passed to the 'moderate' stage, the presence or incidence of gross vascular degeneration will cause rapid progress of the neurotic degeneration with gross dementia."

In some general remarks on the pathology of dementia it is stated that "the morbid appearances are the physiological results of the loss of cerebral substance caused by the neuronic degeneration, which is the physical expression of dementia reacting on the mechanical conditions existing within the skull-cap." The skull being a closed bony chamber, the replacement of lost cerebral tissues is affected by hypertrophy of the membranes, excess of fluid, &c. Non-traumatic subdural deposits are similarly explained. In this connection the effects of gravity on the intracranial contents of the cadaver are gone into, and the results of a series of very interesting experiments on twenty skulls given. It was found that the blood content of the cranium at once decreases by about 22 cc. when the skull-cap and dura are opened and gravity is permitted to act, this blood passing back from the veins and sinuses into the general venous system.

In the final portion of Part I. several pages are intercalated on the etiology of general paralysis, and the influence of syphilis and stress of life are emphasised; this disease the author holds to be "a progressive dementia occurring in syphilised, potential, or actual lunatics possessing the type of hereditary insufficiency of the cortical neurones which is prone to result in premature degeneration."

Part II.—Definitions of the terms amentia and dementia are given. Under the heading of amentia are included such divergent types as idiocy, paranoia, recurrent insanity, and hysteria, and it is interesting to compare this conception of amentia with that expressed in Tredgold's paper; the author, however, proceeds to make out that an under-development of the pyramidal layer of nerve-cells brings all such cases under one category.

The histological portion of the paper is now reached. It consists of a great number of micrometric measurements of the depths of the cell-laminæ in a selected portion of the cortex, and in short resolves itself into the conclusion that a micrometric examination of a small area of prefrontal cortex will suffice to enable one to form a judgment on the degree of pre-existing dementia.

Twenty cases were utilised; these included normal brains,

those of normal aments, imbeciles, cases of chronic insanity without dementia, and cases showing various degrees of dementia; and in all cases a portion of cortex from the anterior pole of the hemisphere was chosen for examination. The various reasons which have influenced the author in the selection of this locality have mainly reference to his previous findings regarding the situation of wasting in dementia, and to his work on the visual area. It is stated that the prefrontal lamination is practically identical with that of the visuo-psychic area: this assertion we must contradict, both cell and fibre appearances are totally dissimilar.

The author concludes that the differences between the several cases exist chiefly in the pyramidal layer of cells; the degree of its development in normal infants and congenital aments varying directly with the mental power of the individual, and the degree of its retrogression in dementia existing in the patient.

While admiring the earnestness and thoroughness of Dr. Bolton's work, we cannot help thinking that the macroscopic changes described constitute uncertain and unstable criteria for forming judgment on subtle degrees of mental disturbance, and his case is marred by one weak point, viz., that he does not take into consideration the influence of bodily disease on the cranial contents. Concerning the histological portion of the papers, which represents an enormous amount of work, we would only add that, while we disapprove of cortical measurements for ordinary purposes, we are satisfied that in Dr. Bolton's hands this method becomes a thoroughly reliable one.

The Pathology and Morbid Histology of Juvenile General Paralysis. By GEORGE A. WATSON, M.B., C.M.Edin. 126 pp.

This paper is founded upon a thorough clinico-pathological examination of twelve cases of juvenile general paralysis, and, from the etiological data given, the conclusions seem justified that the juvenile general paralytic comes frequently of a prolific but degenerate stock; that syphilis plays an essential part in the genesis of the disease, and that as the juvenile is less affected by subsidiary factors, such as stress, alchohol, &c., he represents a purer type of disease than the adult.

The most important microscopic changes found are those in the nerve-cells which are discussed under two headings—(1) acute; (2) chronic. The chronic alterations consist of chroma-

tolysis usually beginning at the base of the cell and gradually leading to more or less complete disintegration of the cytoplasm; this change is associated with relatively slight affection of the nucleus, which may persist even after practically total disintegration of the cell-body. Another striking feature is the general absence of pigment in the diseased cells, making it appear that the development of this substance is a question of age rather than degeneration.

Stress is laid on two varieties of acute changes—(1) swelling of the cell and chromatolysis, with occasional vacuolation; (2) coagulation necrosis. The former consists essentially of a curious balloon-like enlargement of the cell-body with chromatolysis beginning at the periphery, coupled with increase in size and dislocation of the nucleus. The change may be seen in all the cortical laminæ and is looked upon as a manifestation of œdema, being best studied in parts so affected, but to us it seems that the condition might also be compared with Marinesco's *réaction à distance*. In coagulation necrosis the cells are commonly shrunken, stain diffusely and intensely with various aniline dyes and the nucleus is obscured. This change is confined to the fairly large and medium-sized pyramidal cells and has been proved by comparison to be identical with that which Dr. Mott observed after experimental occlusion of cerebral arteries, hence the author concludes that coagulation necrosis is to be associated with the circulating disturbances, that is, the stasis, so common and often so intense in certain positions in this disease, a conclusion with which we entirely agree. As a result of the discovery of these changes in the nerve-cells it is, assumed that "a slow atrophic degeneration is the primary and essential change as far as the nerve-cell is concerned in general paralysis, but frequently large numbers of nerve-cells are destroyed by a more acute or subacute process."

In discussing the condition of the cortical nerve-fibres appropriate allusion is made to the danger of drawing conclusions from imperfectly stained specimens, and to the difficulty in demonstrating the fibres in the general paralytic cortex; acute degeneration was always to be observed in the radiary projection bundles, but not in other cortical systems, and just as was the case with the acute cell changes so also the nerve-fibre degeneration was most profound in the region of vascular disturbance; there was also abundant evidence of chronic degeneration of the fibres, but the conditions here were reversed, the radial fibres being invariably less involved than the horizontal ones. The fibre atrophy is stated to

have been patchy, one gyrus being healthy, while the next was extensively degenerated; but we might indicate that in the normal brain two neighbouring gyri may show an entirely different fibre arrangement, hence we cannot attach much importance to a statement of this kind without more orientation details, in spite of the plausible explanation that the patchy atrophy may be due to vascular changes.

In considering the relationship between the vascular changes and neuronic degeneration, the statement is made "that the chronic degenerative changes affecting the neurons are not proportional to the amount of chronic structural change in the vessel walls, whilst the acute changes are proportional to the amount of vascular congestion, and of active proliferation of the elements in and about the vessel walls."

A clear account of the development of the neuroglia cells is given, but the author is unable to give an unmodified subscription to the general statement that the amount of neuroglia increase is an index of the extent of the neuronic degeneration.

In an estimation of the order of affection of the cortical areas from the various changes mentioned above the prefrontal convolutions are given pride of place, the superior and inferior parietal lobules and the upper temporal gyri come next, then Broca's convolution and the posterior portion of the second frontal, then the central gyri, particularly the post-central, and last of all, the occipital. But, in assigning such a topography to the distribution of these changes, Dr. Watson is alive to the fact that he is treading on dangerous ground, and is mindful of the risk of drawing conclusions from an examination of portions of the cortex only.

In conclusion, Dr. Watson points out that it has been his endeavour to give a description and exposition of his own findings and deductions rather than a criticism of the views of others, to which we might add that the research is a valuable contribution from the hands of an extremely painstaking and accomplished histologist.

The Coagulation-Temperature of Cell-Globulin, and its Bearing on Hyperpyrexia. By W. D. Halliburton, M.D., F.R.S., and F. W. Mott, M.D., F.R.S.

A short paper of eight pages in confirmation of the hypothesis that the physico-chemical cause of death from hyperpyrexia is due to the coagulation of a proteid which can be proved by

fractional heat-coagulation to exist in protoplasmic structures, and is termed by the authors cell-globulin.

When this constituent of cell-protoplasm is coagulated the protoplasm as such is destroyed, and the temperature at which such coagulation is most readily produced is 47° C., but a more prolonged exposure at a temperature of 42° C. has the same effect. These chemical changes in the brain substance were demonstrated by keeping saline extracts of that tissue, or the "surviving" brain of animals just decapitated, at the prescribed temperature. In brains so treated the chromolytic change in the nerve-cells described by Marinesco under the name "coagulation-necrosis" invariably occurs, and the observers now show that this change is directly dependent upon the coagulation-temperature of cell-globulin. Besides the nerve-cells many other cells of the body are probably affected by a high temperature in a corresponding manner.

The Prevention of Dysentery in the London County Asylums. By F. W. Mott, M.D., F.R.S. 32 pp.

Dr. Mott's well-known views on dysentery and its causation are repeated, and his conviction that a system of notification and isolation, a strict observance of principles of disinfection and an improvement of sanitary and hygienic conditions would lead to its suppression, is supported by reliable *post-mortem* evidence showing a pronounced fall in the prevalence of the disease at Claybury Asylum, where such measures have been vigorously carried out. Further instances of direct infection from patient to patient are recorded, and rules for the guidance of the asylum staff in preventing infection specified, but otherwise the paper contains little that has not appeared in Dr. Mott's previous publications.

The Range of Immediate Association and Memory in Normal and Pathological Individuals. By W. C. Smith, M.A.Edin., Ph.D.Leipzig, Lecturer on Psycho-Physics, King's College. 39 pp.

This paper consists of an investigation upon the range of immediate association and memory based on a series of experiments, the subjects of which were normal individuals of varying education and intellectual standard, and also selected cases of general paralysis and alcoholic dementia. The method was similar

to that formerly employed by Galton, and is as follows: Series, of varying length, of type-written alphabetical letters were placed before the subject, and read aloud by him, a metronome being employed to ensure uniformity of time in presentation; some seconds later the subject was asked to repeat the letters. The number of letters varied from four to ten, and the reagent's answers were recorded and valued according to the kind and amount of error, *e.g.*, error of transposition, excess, defect, repetition, &c.

After arranging the experiments in groups and presenting them in tabular form, the author concludes that (1) the method is a trustworthy one; (2) in normal subjects the range of immediate memory has usually a definite limit which lies at or about five letters; (3) in abnormal cases the power of reproduction is diminished; (4) in both normal and abnormal cases, within certain limits, the total of errors remains relatively constant, and reaches approximately the same absolute magnitude; (5) none of the groups improved with practice; (6) the method can be used to differentiate between more permanent memory and the power of immediate reproduction.

The tables are very interesting; the subject is worthy of much more attention than it has hitherto received, and we shall look forward with interest to the completion of the research on recognition which the author mentions.

Pathological Changes in the Medulla Oblongata in Acute Diphtheric Toxæmia. By CHARLES BOLTON, B.Sc., M.D., M.R.C.P. 29 pp.

Dr. Charles Bolton's contribution is based upon the systematic examination of a series of thirteen cases dying during the acute toxic stage of diphtheria, eleven of which succumbed to progressive heart failure due to the severity of the toxæmia, and two to asphyxia, due to involvement of the respiratory passages, the latter being chosen for purposes of comparison, as the toxæmia was less severe.

In an endeavour to trace the exact pathology of progressive heart failure in the acute toxæmic stage of diphtheria the whole medulla oblongata, along with the vagus nerve and heart, were exhaustively examined. In those nuclei of the medulla oblongata which are composed of the smaller variety of nerve-cells, including the sensory nucleus of the vagus, scattered degeneration was present. The nuclei composed of large multipolar cells, on the

other hand, were quite normal, with the exception of the nucleus ambiguus and nucleus centralis inferior, and these two nuclei showed degenerative alterations so marked as to suggest that the cardiac fibres of the vagus originate from them. The amount of degeneration in each case seemed to be proportionate to the degree of the toxæmia, but with all these cell changes it was remarkable how the vagus nerve itself remained healthy, and the author's explanation that it was due to the fact that all the cases died at an early stage of the disease does not seem wholly applicable, because we notice that one lived to the thirteenth day. The heart muscle in the more toxic cases showed marked fatty degeneration, as well as cloudy swelling; in less toxic cases, the latter condition alone was found.

It is supposed that all the above-mentioned changes may occur within the first few days of the disease, and the degree of degeneration in the medulla runs *pari passu* with that in the heart, hence the irregular pulse and fatal syncope of acute diphtheric toxæmia are probably due to the disturbed innervation of an acutely degenerated heart; while the cardiac dilatation is the result of weakness of the heart-wall.

Note upon the Choline Test for Active Degeneration of the Nervous System. By F. W. Mott, M.D., F.R.S. 6 pp.

The *modus operandi* of this test for the presence of choline in the blood has already been made known in papers published by Dr. Mott and Professor Halliburton, and it is again described in order that its obvious simplicity may encourage others to give it a fair trial. Since the presence of choline is indicative of acute degeneration, the test is of course only applicable when the organic changes are active, but it might, for instance, be employed to verify the extension of mischief in an old lesion; and used quantitatively, the amount of destruction in cases of softening, hæmorrhage, and kindred conditions, might possibly be guaged.

In conjunction with others, Dr. Mott has hitherto obtained positive evidence of the destruction of nervous tissue in three cases of beri-beri, one of alcoholic neuritis, one of combined sclerosis, one of Pott's disease, two of tabo-paralysis, three of disseminated sclerosis, two of amyotrophic lateral sclerosis, and one of focal myelitis.

Alfred W. Campbell.

Handbuch der pathologischen Anatomie des Nervensystems. Edited by Dr. E. FLATAU, Dr. L. JACOBSON and Dr. L. MINOR. (Published by Williams & Norgate, 14, Henrietta Street, Covent Garden, London).

THIS book, of which the first volume alone is at present published, is the joint work of well-known writers who contribute articles on subjects in which they are especially versed under the editorship of Dr. Flatau, Dr. Jacobson, and Dr. Minor.

The first part of the work deals with the methods for examination of the nervous system.

Four subdivisions are made in this chapter—in the first is given a somewhat detailed description of the best method for removal of the brain and spinal cord from the body; in the second is given the technique of microscopical investigation of the nervous system; in the third, the bacteriological examination of the nervous system; and in the fourth a useful section on the changes found in the nervous system which are not of a pathological nature.

This last sub-division is again divided into firstly, changes occurring in tissues during life, and, secondly, changes due to *post-mortem* conditions.

Under the first heading come the variations met with during development, those due to physiological use, such as the changes found in the chromophilic substance of the nerve-cell after work; and the changes met with in the posterior columns after prolonged exertion. These latter are on the border line between physiological and pathological conditions.

The changes in the tissues due to stasis of blood at the time of death and the changes due to senility are pointed out. The possible fallacies of the Marchi method are discussed, and the alteration which the nerve-fibre undergoes owing to *post-mortem* changes.

The changes produced by injury to the cord, such as heterotopia, during its removal from the body, are dealt with, and the view that they are the result of *post-mortem* injury is accepted.

The senile changes in the nerve-cells of the brain and spine are described. The pigment increases in amount and diminishes the nutritive capacity of the cell. The number of cell processes become fewer and the finer processes disappear. The cell becomes small and may eventally completely atrophy.

Two kinds of pigment are described, the one yellow in colour

and the other dark brown; the former would, by its dark staining with osmic acid, seem allied to the fats, while the latter is a true pigment.

Having thus dealt with the method of examination and possible anatomical variations of the nervous system, the next chapter, written by Van Gehuchten, describes the appearance of the normal nerve-cell and the changes which take place under pathological conditions. The normal cell varies with regard to its staining reaction under different functional conditions. A cell is said to be in a pyknomorphic condition, if the chromophilic substance is very rich and closely packed, so that the cell protoplasm is deeply stained, while it is said to be in an apyknomorphic condition when the chromophilic substance is scattered. A cell in a condition intermediate between the above is said to be parapyknomorphic.

The pyknomorphic condition of the cell probably indicates that the cell has been at rest, while the parapyknomorphic and apyknomorphic condition indicates functional activity and exhaustion of the cell.

The alteration of the shape of the cell and the changes found after lesions of the axis cylinder, firstly, of the peripheral neuron, whether motor or sensory; and, secondly, of the central neuron, whether motor or sensory, are described and figured. The changes due to circulatory disturbance, to inanition, to slow and rapid asphyxia, to hyper- and hypo-pyrexia, and to various forms of poisons, are described. The changes met with in uræmia, glycosuria, tetanus, and other allied conditions are dealt with.

The significance of chromolysis, of pigmentation, of changes in the achromophilic substance, of fissuring of the protoplasm, vacuolisation and changes in the nucleus are all fully dealt with.

The chapter on the general pathological anatomy of the nerve-fibre is written by Dr. Lugaro.

The rate at which nerve-fibres of different physiological functions degenerate varies, and the sensory fibres degenerate more rapidly than the motor.

In the central nervous system the same holds good, and secondary degeneration of the posterior column is more rapid than that of the direct cerebellar tracts, and both of these tracts degenerate earlier than the pyramidal tracts.

The rate of degeneration varies also in different species; for instance, degeneration in a winter frog's nerve after section is thirty times slower than that in a rabbit's nerve.

The difficult question of regeneration of nerve-fibre after injury and section is fully discussed.

An interesting discussion on so-called retrograde degeneration concludes this part of the work.

Five groups are distinguished. The first an ascending neuritis starting in the small end branches of the nerve and spreading up the nerve as a true interstitial inflammation.

The second an acute degeneration of certain fibres of the central end of the nerve after section.

The third a slow degeneration of nerve-fibres of the central end of the nerve following amputation.

The fourth a degeneration of nerve-fibre occurring in new born children after injury.

The fifth in which, after a variety of lesions, degeneration takes place in the posterior columns of the cord.

After fully discussing the subject the conclusion arrived at is that the Wallerian law still holds good and that the above conditions are all explicable by the view that there is a lesion of the trophic centre of the fibre involved.

The following chapter by the same author deals with the pathology of neuroglia.

Possibly the most important chapter in this volume is that by Nonne and Hans Luce dealing with the pathological changes of the vessels.

A good description of the normal appearance of the vessels of the nervous system is first given followed by the description of the changes found in arterial sclerosis, atheromatous degeneration, and the formation of miliary aneurism in the brain.

Syphilitic endarteritis is next described and numerous illustrations given of the effect produced by the obstruction and blocking of those vessels. Hæmorrhage occurs much less frequently from a vessel affected with syphilitic endarteritis than from a vessel with atheromatous degeneration. Tubercle of the wall of the vessel, the condition of the vessels in acute anterior poliomyelitis, and in myelitis are described, and the part played by the condition of the vessels in production of combined degeneration of the cord, in multiple sclerosis, in chronic myelitis, in syringo-myelia and tabes dorsalis are all discussed at length. A fairly complete bibliography is given at the end of the various sections.

The first volume closes with a chapter by Stroebe on the changes taking place in the skull and membranes of the brain. The work is most complete and as a book of reference will be invaluable to the worker in neuropathology, for not only will he find many points described in this volume which can only have

been derived from the personal experience of the writer, but also by the convenient manner in which the bibliography is arranged he will readily obtain reference to papers or subjects allied to those which he is investigating.

FREDERICK E. BATTEN.

The Mind of Man; a text-book of Psychology. By GUSTAV SPILLER. (Demy 8vo. pp. 552—xiv. London: Sonnenchein, 1902.)

THE first question that a reader should ask in opening a new work on psychology is—How far is it intelligible? In psychology, obscurity has of late years passed for profundity, and unintelligibility has has been so habitually offered—and accepted—as a mark of genius, that clearness of expression is no longer striven for, is no more aspired to, is indeed become discredited. Exactness of thought, clearness of expression, precision of language, these are looked upon as signs of shallowness and poverty of mind; and he alone whose meaning can be differently interpreted by every different reader, or whose meaning cannot be fathomed at all, is regarded as having a meaning worth considering. Every raw undergraduate who has attended a course of lectures and read his first misty text-book, has a cheap sneer for Herbert Spencer, whom he cannot fail to understand; and joins in the worship of Hegel, whose meaning no one ever yet was sure of.

So that, as aforesaid, upon opening a new psychological work, the reader's first enquiry should be how far it is intelligible, for upon this quality his judgment of the book will depend. If he is of the prevailing school, and desires to be in the forefront of psychological fashion, then, in proportion as the book is imperfectly thought out, ill-written and obscure, in that proportion will he be loud in its praise and pile up laudatory superlatives; while, if he is one of the discredited few who still maintain that obscurity of meaning and slovenliness of diction imply fogginess of thought and inefficient analysis, then, equally, he will judge of the book by its intelligibility, and will say that, if it is readily intelligible, its author can at least think vigorously and express himself clearly, while if it is intelligible with difficulty, he will regard this difficulty as a capital defect.

If we construct a scale of intelligibility along the range of the

alphabet, we should place at A such writers as Hobbes and Spencer, whose meaning is always transparently clear; while at Z, the zero of intelligibility, we should place Hegel, the study of whom is like exploring a coal-mine on a foggy night. On such a scale Mr. Spiller would be placed about W or X. He is not always wholly meaningless, but his meaning can usually be attained only by labour which is greater than the meaning is worth.

According to the preface, Mr. Spiller has initiated an entirely new method in psychology. For the first time in history, psychology is become scientific by being studied upon the scientific, *i.e.*, the experimental, method. "Its most pervading feature is, perhaps, the organic conception of the life of thought and action which the enquiry has forced into the foreground." Here, at the very outset, I find a statement which is evidently intended to be of the highest importance, but is quite unintelligible to me. I do not know what an "organic conception" is, nor how it differs from an inorganic conception, if there is such a thing; nor do I know what is meant by the "life of thought," nor the "life of action." Mr. Spiller continues: "Hence the current notions as regards motives, pleasure-pain, reason, attention, association, habit, and the will, which suggest no intricate and developed organic processes, have either been rejected or considerably modified. Thus also the tripartite division into intellect, feeling, and volition, has been replaced by a close analysis of the nature and satisfaction of needs or functional tendencies. The key to the whole work is contained in chapters vii., ii., and iii." So far as I am able to interpret this passage, which appears to be the "key to the whole work," it would seem that because the most pervading feature of the book is perhaps the organic conception of the life of thought and action, therefore "current notions as regards" motives and other things have been rejected or modified, and therefore also the tripartite division of something unmentioned has been replaced by a close analysis of something else.

Examination of the preface reveals, it is clear, a high degree of unintelligibility, and consequently we are prepared to class the book as extremely profound, or extremely confused, according to the psychological school to which we happen to belong. Not to judge the book entirely by its preface, let us turn to the chapter singled out by the author as containing the key to the whole work and see if it leads to any different conclusion.

Chapter vii. is called "Systems as Need-determined," and we naturally ask what Mr. Spiller means by a system, for he appears

to use the term in a peculiar sense. On p. 40 we find the explanation, "*Nothing less than a total reconstruction of human language can satisfy rational demands*" (italics in original). Such a revolution, Mr. Spiller sees, must be the work of centuries, and not of a solitary individual; but still he makes a beginning, and his first essay in the reconstruction of human language deals with this very word "system." "System," according to Mr. Spiller, means "anything given whatever." It is scarcely fair to his readers to give his first definition of a reconstructed term in language that is already reconstructed. "Anything given whatever" may be reconstructed human language, but it is not unreconstructed English. As far as can be gathered from the context the word "given" in this definition is superfluous, and is inserted on the ground of reconstruction only. Throughout the book, "system" appears to mean "anything whatever," and whether given or taken, to whom given or from whom taken, is wholly immaterial.

Chapter vii. contains seventeen sections, every one of which deals with some aspect of volition, and we gather from this content of the chapter that its title, "Systems as Need-determined," has something to do with the will; but in drawing this inference we reckoned without the reconstructed language which Mr. Spiller uses. His definition of *change in systems as need-determined*, given on p. 42, is:—[Need-determined change or functional adjustment. Thinking; acting.]

The first of the seventeen sections in chapter vii. contains sixteen numbered propositions, of which the following are samples:—(1) When I will to move my eyes, limbs, or body, it is the action most natural to those parts which results. (10) I let articulate sounds, regardless of sense, freely escape my lips. (Here follow a number of disconnected words). (12) I will to move my hand to the right; then I will to move it to the left. In each case the act of willing is not willed. Will, then, does not explain itself; it itself develops out of a previous system (see definition of "system." Reviewer.) (14) As a matter of information we say "yes" or "no." Or, after weighing an argument, looking at a drawing, deliberating over an action, we say "yes" or "no" in answer to a question. We do not affirm or negative every statement which flits by. It is the same with the will."

These sections have been selected solely on account of their brevity; they are neither less nor more luminous than the remainder. To say that they throw no light upon the problem of volition would be beside the mark altogether. It would be equally

relevant to say that the X-rays throw no light upon the Norman Conquest. Not only do they not contain any inkling of the solution, but they have not any bearing upon even the statement of the problem. They lead to pure bewilderment and amazement. They make us wonder what the writer is driving at. The words seem to be connected in some sort of order, as if they ought to make sense if one could only understand them aright; but the more one puzzles over them the more hopelessly muddled one gets.

The disadvantage of using a reconstructed language, even though the reconstruction is but partial, is very conspicuous in other parts of Mr. Spiller's book. Thus on p. 165 he defines abstraction thus:—"When a certain aspect of an object is singled out for inspection, we are said to abstract. Thus we may be thinking of the hands of a man, and neglect the man of whom they are a part." Anyone who did not know that Mr. Spiller was using reconstructed language would conclude from this passage that he did not know the meaning of the term abstraction, especially as he says that "The fact of abstraction, as usually understood, is of no psychological importance." It is evident that he is using the term abstraction in a Spillerian sense, and does not mean the process ordinarily so called, which lies at the root of the whole process of thought, and without which thinking is impossible. In a recent publication I had to deplore the fact that in psychology anyone may call anything by any name that he pleases; I must admit that I was not then aware of such a complete exemplification of the dictum.

CHAS. MERCIER.

Maladies Nerveuses, Diagnostic, Traitement. (*Nervous Diseases, Diagnosis and Treatment.*) By Dr. J. VIRES. With Preface by Prof. F. RAYMOND. Montpellier, Coulet et Fils. 1902.)

THE object of this work is to furnish a manual of neurotherapeutics. The system adopted by the author is to give a brief outline of the main clinical features of each disease before discussing the treatment. In some instances tables of differential diagnosis are given, as in the case of meningitis and typhoid, hysterical and organic paralyses, &c. A short account of etiology

is added, and then comes a full description of the treatment to be followed, and the methods of dealing with complications and clinical varieties of the disease under consideration. There is much useful information to be gained from perusal of Prof. Vires's work, and, speaking generally, the treatment is sound. The writer gives a larger number of prescriptions than is perhaps necessary. The chapters relating to such subjects as Neurasthenia, Epilepsy, and Hysteria are the best in the work, some of the others being unduly drawn out. The chief objection to the book, in our opinion, is that the diseases are arranged alphabetically. It may to some extent be a convenience in turning up some special subject, but this does not make up for the disconnected style of the work.

DOUGLAS STANLEY.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

A Meeting of the Society was held in Edinburgh on Saturday, June 27, 1903.

Physiological and Pathological Demonstration.

(1) Dr. Wakelin Barratt.

Microscopic specimens of the nervous system in rabies.

(2) Dr. Byrom Bramwell.

Series of photographs illustrative of morbid clinical appearances.

Series of specimens, photographs, and drawings, illustrative of morbid pathological appearances.

(3) Dr. Edwin Bramwell.

Sections and photographs of spinal cord from a case of early tabes.

Sections from a case of pressure paraplegia.

Photographs showing degeneration of the septo-marginal tract.

Sections of brain and spinal cord showing bilateral pyramidal degeneration.

Sections of Professor Edinger's case of unilateral atrophy of the cerebellum. The specimen prepared by Professor Weigert.

Photographs of clinical cases.

(4) Dr. Alexander Bruce.

Hæmorrhage into spinal cord; macroscopic and microscopic preparations.

Tumour pressing on cord.

Syringomyelia; macroscopic and microscopic preparations.

Tumour of lower end of cord.

Tubercular tumour of pons, destroying sixth nucleus.

Three fibro-cellular tumours, growing from dura and pressing on side of pons.

Spinal cord bony plates of arachnoid.

Hæmorrhagic tumour of pons.

Two cases of hypertrophy of pons.

Unilateral glioma of pons.

Two cases of cerebellar hæmorrhage.

Hæmorrhage into pons.

Aneurism of anterior-cerebral artery.

Shoulder-joints from locomotor ataxia.

Case of porencephaly (slide).

Sections of cord after amputation of parts of lower extremities.

Photographs of achondroplasia.

Microtome for cutting large sections with spirit.

(5) Dr. R. A. Fleming.

Sketches (microscopic) of regeneration in nerves after section, and in several cases of alcoholic peripheral neuritis.

Sketches and photographs of hæmorrhage into the intersheath spaces of the optic nerve in cases of subarachnoid hæmorrhage, causing retinal hæmorrhages in the corresponding eye.

Sketches and microscopical demonstration of neuroglial and vascular changes in the optic nerve in cases of optic neuritis and optic atrophy following on intracranial tumour.

Specimens.

(*a*) Aneurism of the circle of Willis.
(*b*) Brain with remarkable atrophy of convolutions.
(*c*) Brain showing results of septic thrombosis in superior longitudinal sinus (erysipelatous).

(6) Dr. G. A. Gibson.

Brain from which a sarcoma was removed eight years before death.

Pictures.

Pathological.

A sarcoma of the frontal lobe.
Sarcoma of frontal lobe.
Cerebellar tumour (deeply situated).
The same in section.
Cerebellar tumour (superficial).
Sinus thrombosis.
Same—deeper section.
Porencephaly—vertex.
,, lateral.
,, base.

Clinical.

Case of facial paralysis with anterior poliomyelitis.
Distribution of pain in angina pectoris.
Series of cases of Graves' disease.
Photographs of Graves' disease showing von Graefe's symptom.
Series of cases of ocular paralysis.
Series of photographs of pseudo-hypertrophic paralysis.
Another series of the same from another patient.
Series of photographs of acromegaly.
Skiagrams of the same.

7 Professor Greenfield.

Various cerebral tumours—microscopic and macroscopic specimens.

(8) Dr. P. T. Herring.

Sections illustrating the spinal origin of the cervical sympathetic.

(9) Dr. Harvey Littlejohn.

Specimens illustrating injuries to the brain and intracranial hæmorrhage.

(10) Dr. W. Page May.

Brain of monkey showing double porencephaly.

(11) Mr. Alexander Miles.

Specimens illustrating middle meningeal hæmorrhage (with stereoscopic photographs).

Specimens illustrating injuries of the brain (with stereoscopic photographs).

Specimens and stereoscopic photographs illustrating spina-bifida.

Specimens and photographs illustrating injuries of the spine.

Specimens and photographs illustrating tuberculous disease of the spine.

Congenital malformation of the brain from a case of spina-bifida.

(12) Dr. Harry Rainy.

Skiagrams and specimens of achondroplasia.

Dr. Harry Rainy and Dr. J. S. Fowler.

Microscopical specimens from a case of congenital nuclei facial diplegia.

Dr. Harry Rainy and Mr. Stiles.

Skiagrams of injections of the cerebral blood-vessels.

(13) Dr. Ford Robertson.

Lantern demonstration illustrating results of histological and experimental investigations into the pathology of general paralysis.

Microscopic demonstration of same.

(14) Professor SCHÄFER.

The effects of section of pyramidal tracts and of anterior columns respectively in monkeys.

(15) Dr. THEODORE SHENNAN.

Specimen of brain showing porencephaly with hydrocephalus.

Spinal cord with calcification in the arachnoid.

(16) Professor SHERRINGTON.

Two spinal reflexes in a dog.

(17) Dr. A. H. H. SINCLAIR.

Fields of vision as taken with small white test objects after the method devised by Bjerrum, with charts showing fields taken in various conditions of disease.

(18) Mr. A. A. SCOT SKIRVING.

Cast and skiagrams of a knee from a case of tabetic arthropathy.

(19) Mr. STILES.

Specimens and casts.

(20) Mr. ALEXIS THOMSON.

Microscopic and macroscopic specimens of neuromata and neuro-fibromata.

(21) Dr. WATERSTON.

Some specimens illustrating the development of convolutions.

Bilateral division of the Rolandic fissure in the brain of a child.

Photographs of the brain in Mongolianism. Specimens from the base of the skull from a case.

Dr. WATERSTON with Dr. DAVID HEPBURN.

Photographs and sections to illustrate the segmental arrangements of the gray matter and the groups of cells in Phocæna com.

Dr. WATERSTON and Dr. EDWIN MATTHEW.

Sections of the spinal cord showing variations in the pyramidal tract, and from a spinal cord of anencephaly.

(22) Dr. CHALMERS WATSON.

Photographs illustrating some clinical features of disease of the nervous system in a dog—a rheumatic subject affected with chronic endocarditis.

Photographs illustrating the *rôle* of vascular changes in chronic disease of the nervous system in horses.

Dr. CHALMERS WATSON and Dr. G. R. WILSON.

Microscopic demonstration on some visceral lesions in two cases of acute insanity. Specimens shown from the gastro-intestinal, respiratory and urinary tracts.

The first part of the meeting was held in the Physiological Laboratory of the University, the President, Prof. LANGLEY being in the chair.

Prof. SHERRINGTON showed a dog in which the eighth cervical segment had been excised and demonstrated the spinal reflexes in this animal.

Dr. A. H. H. SINCLAIR showed a method for investigating the fields of vision with small white test objects. The method had been devised by Bjerrum.

Dr. W. FORD ROBERTSON gave a lantern demonstration illustrating the results of bacteriological, histological, and experimental investigations into the pathology of general paralysis. He stated that the investigation was by no means complete but that he put forth the matter more as a hypothesis than as a proved conclusion. The points specially illustrated were the occurrence of a bacillus resembling the Klebs-Löffler bacillus in the catarrhal exudations in the respiratory and alimentary tracts and the pathological changes produced, especially in the nervous system, in rats by feeding with pure cultures of this organism. These changes were in certain respects of the same character as those that occurred in general paralysis.

Dr. Clouston, Dr. Henry Head, Dr. Byrom Bramwell, Dr. Hudson Bury, Dr. Hyslop and Dr. Barratt took part in the discussion.

Dr. ALEXANDER BRUCE exhibited, by means of the lantern sections of the spinal cord to show the localisation of function

in the lumbo-sacral part of the cord. In one case of amputation at the knee the nuclei supplying the muscles below the knee had undergone degeneration. In the second case a rare opportunity was afforded of localising the centres in the lumbo-sacral part of the cord. It was a case of amputation at the hip-joint on account of tuberculosis and practically all the muscles attached to the os innominatum were removed, only the deep muscles being left (the psoas, the iliacus, and the external rotators of the hip). The first case demonstrated that the muscles below the knee were almost entirely supplied by the postero-lateral and post-postero-lateral groups of nerve-cells in the lumbo-sacral part of the cord. In the second case degeneration of the nerve-cells had begun as high up as the second lumbar segment. At the level of the third lumbar segment only three or four cells were seen in the section to be intact and at the level of the fourth segment the whole of the nerve-cells showed Nissl's "reaction at a distance." Much the same appearances continued throughout the sacral segments of the cord.

Dr. R. A. Fleming gave a lantern demonstration of the neuroglial and vascular changes in the optic nerve in cases of optic neuritis and optic atrophy following on intracranial tumour.

Dr. D. Waterston and Dr. D. Hepburn gave a lantern demonstration of photographs of sections of the spinal cord of the porpoise (phocæna communis). They stated that the investigation had been undertaken in order to determine the effect which was produced upon the shape of the gray matter of the cord and on the groups of motor cells in a mammal with such altered musculature as one of the cetacea. The fore-limbs were rudimentary and the hind-limbs were entirely absent. It had been found that the different segments of the cord had characters by which each could be recognised from any other. The groups of cells were pointed out in the cervical region and it was shown that in the lumbo-sacral region there was an absence of lateral groups of cells such as were found in man. There was marked lumbar enlargement in which the anterior horns of gray matter were elongated and contained deeply placed nuclei for a special nerve supplying abdominal and perineal muscles. A condition of heterotopia of the gray matter in the posterior columns was also demonstrated. On the whole the investigation corroborated views which had been recently brought forward as to the function of some groups of cells in the human spinal cord.

The second part of the meeting was held in the Royal Infirmary and was devoted to the exhibition of Clinical cases.

Dr. CLOUSTON demonstrated three cases of exophthalmic goitre to illustrate the mental condition often found in advanced cases of this disease. All three patients were affected with ideas of persecution and of grandeur. The first case was that of a woman, aged about 35 years, who had been under treatment in the medical wards for a considerable time and who had at last been transferred to the asylum. She had ideas of poison in her food as well as those of wealth, her sensations being generally of a morbid nature. The toxic influence of this disease was similar to the action of alcohol on the brain. The second case was that of an oldish woman. In her case the thyroid gland was only slightly enlarged but she had had a hard life, had suffered from many diseases, and had undergone almost every influence which caused nerve depression. She had hallucinations—ideas of expansion as well as perversions of ordinary sensations. In all Dr. Clouston's large experience of giving thyroid gland substance to patients he had never found that it produced symptoms at all like those of exophthalmic goitre. The third case was also in an oldish woman and the disease had persisted for four years. It had commenced in acute melancholia and now she also had delusions of persecution and grandeur. The last two patients, though imagining that they were being poisoned, yet took their food and were well nourished, and, indeed, fat.

Dr. BYROM BRAMWELL showed: (1) A case of removal of a cerebral tumour. The case was that of a man whose disease began in 1899 in what was considered an epileptic fit. Attacks of Jacksonian epilepsy then developed which began in the left foot. There was also localised tenderness over the right motor area of the cerebrum and the patient became emaciated. There were no other symptoms. Mercury and iodide of potassium were administered vigorously. Mr. J. M. Cotterill saw the patient in consultation and an operation was decided on. On opening the calvaria a tumour of the size of a Tangerine orange was found attached to the meninges of the brain. It was a sarcoma and was removed. The patient suffered very severely from shock and, in fact, nearly lost his life. An excellent recovery was, however, made and at the present time he states that he never felt better in his life, although there is still a little weakness in the left leg and foot. No part of the brain substance was removed. (2) A case of tabes with optic atrophy. Benedickt

had stated that when optic atrophy occurred in the course of this disease the affection soon became arrested. If this were so this case was exceptional. The patient was a man, aged 34 years. When first seen five years ago he had all the signs of pre-ataxic tabes. Two years previously to this, however, he had become blind from optic atrophy and the pupils were dilated. The patient remained absolutely well until eighteen months ago when lightning pains commenced in his legs, and six months later ataxia began to develop. At present the ataxia was so marked that he could not stand. Such a case absolutely disproved the dogmatic statement of the arrest of the disease when optic atrophy showed itself primarily. Dr. Bramwell had observed a number of similar progressions of this disease. The next two cases were illustrative of hæmorrhage into the spinal cord. (3) A boy who in September, 1901, was suddenly seized with paralysis in jumping off a car. At first it was the left leg which was paralysed completely, but it soon extended to the left arm and then to the right arm and leg. The onset was absolutely sudden, as the boy was in perfect health up to the moment of jumping off the car. After the first few days there was no tactile defect. The perception of heat and cold was, however, markedly perverted and this had persisted ever since. He was very ill at first and the bladder had to be opened on account of hæmorrhage taking place into it. Bed-sores formed and he developed a septic temperature. Eventually he recovered, but the defect of sensation was marked. The localisation of touch was perfect all over the body, as was also that of pain, but from the level of the nipples downwards to the toes he confounded the sensations of heat and cold. The case had evidently been due to hæmorrhage into the central region of the spinal cord and was probably into a syringomyelia. (4) A man, aged 44 years, who, though previously quite well, suddenly and completely lost the power in his legs without feeling any pain in his back. There was also marked paralysis of the arms and the hands remained in the position of tetany. He also suffered from cramps. The localisation of touch and pain was perfect, but here also there was a marked alteration in the temperature sense. He made an excellent recovery, but from the level of the nipples downwards there were analgesia and loss of temperature sense. The lesion was also probably a hæmorrhage into a latent syringomyelia. (5) A man who had been under observation for seven years suffering from extremely severe gastric crises which recurred once in every six or seven weeks and each lasted from ten to fourteen days. There was no ataxia, but the Argyll-

Robertson phenomena, lightning pains, and difficulty in micturition were present. Marked hypotonia was present and his movements were acrobatic. The patient was being treated with X-rays to the back and with remarkable benefit. Dr. Bramwell had seen good results from this light treatment in two other cases. The improvement might, however, be only psychical.

Dr. G. A. Gibson showed a man, aged 68 years, who was suddenly seized with disturbance of vision. The patient had been absolutely steady as regards alcohol until one day when doing hard work he took two glasses of whisky and the next day four glasses. The following day he complained of a general dimness of vision and found that the half of every object was blotted out. There was complete right-sided homonymous hemianopia. There was no change in ordinary sensibility or in the senses. He was, however, on the verge of word-blindness and could only read large type and by aiding himself by his muscular sense in forming the letters with his fingers. At the outset of his trouble he suffered from disturbance of his memory, forgetting his name and place of abode. The lesion was either cortical or more probably subcortical.

Dr. John Thomson showed: (1) A boy, aged $3\frac{1}{2}$ years, who had suffered from birth from lateral head-rotating and conjugate nystagmus. His gait was cerebellar and he tended to fall backwards. He was, however, quite intelligent and began to speak when two years of age. (2) A boy, aged 6 years, who exhibited a condition not previously described. There was a fine general tremor of most of the voluntary muscles on the slightest exertion. On extending the arms and protruding the tongue there was a constant fine tremor very noticeable on laying the hands over the muscles. There was no sensory change and no rigidity. His intelligence was above the normal and the condition was noticed soon after birth.

Dr. R. A. Lundie exhibited a rare case of infantile oculofacial palsy (Gowers) in a woman aged 54 years. The affection was nuclear in origin and affected some or all of the ocular muscles as well as those of the face. There was nothing in the woman's family history to account for the condition. She had a squint which was operated on when she was of the age of 5 years; this might have been a paralytic squint. Her parents stated that she had natural movements of the face as a little child, but so long as she herself could remember she had had no power in the facial muscles. For twenty-eight years at least she had had a left-sided squint, and twenty-one years ago an operation was performed on

her right eye. Since this operation the patient dated her loss of power of the lateral movements of the eye. Her present condition showed that the face was flattened out; though the movements of the tongue were perfect those of the lips were absent. She had a considerable vertical movement of the right eye but less in the left. The lateral movements were lost in both eyes. The iris reacted to light but not to accommodation. The levatores palpebrarum acted to a very slight degree as did also the facial muscles. The paralysis was not absolute. The corneæ were protected by the eyeballs being rolled up under the eyelid.

Professor W. S. GREENFIELD exhibited a boy, aged 18 years, who was probably the subject of a pituitary tumour. He had been absolutely healthy until February 7, 1903, when he was suddenly seized with intense frontal headache and vomiting with loss of memory. These symptoms were most severe during the mornings and persisted for several weeks. He was admitted to the infirmary on March 12, when his condition showed a marked thickening of the lips, nose, and face generally. The hands and feet were full and thick. Marked torpor was present and he slept for many hours consecutively. There were a swelling of the glands and tenderness on the left side of the neck. Mastoid disease was absent. The facies and general appearance led to a diagnosis of acute acromegaly, probably due to disease of the pituitary body. At present there was double optic neuritis of intensity, associated with hæmorrhages and fine degeneration around a yellow spot; slight paresis of some of the ocular muscles, as the sixth, was evident. Sugar was present in the urine (from three to four grains per ounce) but there was no polyuria. The boy's general condition had undergone great improvement during the administration of pituitary substance, which was commenced a month ago, two grains twice, and latterly thrice, daily. His headache was not now complained of and the thickening of the subcutaneous tissues had lessened.

Dr. FLEMING exhibited a girl, aged 18 years, who was admitted to hospital in April, 1902, suffering from marked compression symptoms. Localising symptoms were almost absent, but pointed most to the left arm centre. As she was evidently dying from compression (hydrocephalic cry, temperature running up, and pulse feeble and rapid) the skull was opened over the right motor area. The membranes bulged much but there was no evidence of tumour. On opening the meninges a large amount of cerebro-spinal fluid escaped. The patient rapidly improved and in two weeks regained consciousness and could use the left

arm and leg. The wound healed, but there was a large bulging filled with cerebro-spinal fluid, and any attempt to apply pressure to this induced an exaggeration of the paralysis. At the time of operation there was slight optic neuritis but this soon passed off and her sight was now normal. During the past week the old wound had commenced to exude cerebro-spinal fluid. There was a tuberculous history on the father's side.

Dr. BRUCE showed a woman suffering mainly from paralysis of the right serratus magnus muscle. She was delivered of a child on November 3, 1902, and two days later was seized with an intense pain over the right shoulder-joint so severe that she could not move the arm. There was loss of power in attempting to raise the arm above the shoulder. The right scapula became remarkably prominent when she held the arm forwards or upwards. There was a hollowing in both supra- and infra-spinous regions of the scapula, showing that the nerves supplying the muscles were also affected.

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EDITOR.